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CHEST EXAMINATION

THE CORRELATION OF PHYSICAL AND X RAY FINDINGS IN DISEASES OF THE LUNG

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Third Edition

With 150 Illustrations



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To
M D T
AND
D R T

<i>First Edition</i>	1943
<i>Second Edition</i>	1945
<i>Third Edition</i>	1948

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FOREWORD

WHILE still a medical student I felt strongly that the next generation would condemn the practice of herding cases of proved pulmonary tuberculosis in out-patient departments. There they would sit in rows among the other patients, coughing up their ultimatum to be rewarded by a bottle of cod liver oil and some liniment. But we did have the opportunity of learning our physical grounds from these unfortunate. To-day I can affirm from my experience as an examiner that serious universities that the weakest part of the candidate's equipment is the recognition and interpretation of physical signs in the lungs. To some extent this may be due to the segregation of tuberculous cases into departments where students do not freely enter, but at all moments the tendency to short-cut the examination by relying on the X-ray film using it as a substitute instead of confirmation of ordinary clinical methods. The teachers, however, are not entirely free from blame particularly in the matter of multiplying labels for the various sounds heard through the stethoscope, the degree of accuracy thereby implied is largely illusory and merely confuses the student. It is one of the many merits of Wang Commander Traill's book that he follows the tradition of my own teachers Dr. Lee and Dr. Samuel West by using as few terms as possible and correlating each with its pathological significance.

This plan is characteristic of the whole work, anatomy, physiology, pathology, symptoms, physical signs and X-ray findings are closely correlated enabling the student or practitioner to build up mental concepts of exactly what is happening inside the chest. Many will be interested to learn how greatly modern X-ray technique has advanced interpretation in skilled hands. Even now we will be surprised to find what helpful deductions from physiological principles they habitually ignore. And all will be relieved to find how grasp of the first principles involved will enable them to form the correct opinion without imposing burdensome details on their memory.

As former teacher I know how valuable a contribution this book makes to the understanding of chest problems while the freshness of its approach will be patent to all.

W. LANGDON BROWN

PREFACE TO THE THIRD EDITION

THIS Third Edition has again been slightly enlarged. Reference has been made to recent work on the broncho-pulmonary segments of the lung, a chapter has been added on military shadowing and the last chapter of the second edition has been replaced by a short description of lung tumours.

To illustrate these additions extra figures and X rays have been included and for some of the X rays here reproduced I wish to express my grateful thanks to Dr. P. T. K. Riley and to Dr. Fawcitt, who has done such excellent work on radiological evidence in hæmophilic iron-ore work.

R. R. TRAIL

PREFACE TO THE FIRST EDITION

THIS book is founded on courses of lectures which it has been the writer's good fortune to give to students and post graduates. They now in their present form in great part the attempts made to answer the difficulties of those students who showed an interest in an approach to examination of the chest founded on knowledge of applied anatomy, physiology and pathology without which it is naturally useless to attempt a reasonable interpretation and correlation of physical and X-ray findings.

Correlation is not so difficult to acquire as many students seem to fear. Much confusion has arisen of late years because of the big divisions made by chest physician specialists on the one hand and chest radiology specialists on the other. More recently however there has been a tendency to combine these branches in the chest physician cum radiologist or the chest physician who likes to read his own X-rays. This is all to the good of the modern student, it tends to bring us back to fundamentals. We find it increasingly easy to go beyond the objective reading of films. We can connect physical signs with definite abnormalities of

shadow and combine both with the changes in normal anatomy and physiology so ably expressed nowadays by the specialized lung pathologist who is we must remember but the first offshoot of that type of learned physician who laid his firm foundation for practice in his earlier post mortem room researches when X rays did not exist.

The first Section of the book is therefore devoted to reminders on those salient points of normal anatomy and physiology which explain the abnormalities of the commoner chest diseases of general practice. Like other points of equal importance they are repeated by reference to other sections.

In the second Section on Applied Pathology we could not the main gross and microscopical changes induced by these diseases and in noting the physical signs and the alterations from the normal postero-anterior film that accompany them in their various stages an attempt is made to correlate all three aspects. A certain amount of detailed description is necessary but this is confined as much as possible to fundamentals even if by this statement it appear to be begging the question.

There has been of late years much discussion on the reading of abnormal films and of necessity so much individual variation in reports that an attempt has been made in some quarters to confine them to objective wording. As already indicated this is rather feeble and we can go further and that we should attempt to arrive at criteria for interpretation using the work of the physician the pathologist and the radiologist as a combined whole. We shall all agree that changes of microscopical detail cannot be expected to reflect themselves in stethoscopic signs and on the usual postero-anterior film. We know that a lobe on the lung surface is no more than about one-quarter of an inch in its longest diameter. Nevertheless it is felt that a knowledge of pathogenesis is fundamental for the student who could correlate his physical and X ray findings even if these are demonstrable only when comparatively gross areas of lung are involved. Thus we know that in phthisis stethoscopic signs are late but we can find a reason why they are late and so why we should look for them and as important physical signs such as lack of movement and note that will precede these stethoscopic signs and so warn us of the presence of a specific pathological change. It may be argued that the prime correlations here detailed are built on prior reasoning even if this be justifiable criticism it will be admitted that they

can give some elements that may combine the still too well defined compartments of the pathologist, the radiologist and the physician. For a mere workable explanation would be better than no basis at all.

Section III sums up the physical findings in the diseases discussed in Section II and gives a scheme of interpretation of stethoscopic findings that may act as basis for diagnosis without X-ray findings. No attempt is made to deal with all normal and abnormal stethoscopic signs but only with such additional auscultatory sounds as can be correlated with underlying pathogenesis. It is taken for granted that the student has listened to large numbers of normal chests and a sufficient number of abnormalities to understand the principles underlying the interpretation of breath sounds and chest sounds. The difference between the prolonged expiratory ratio of emphysema and the high pitch faint loud wheeze that denotes bronchial breathing.

It will be seen that all these sections are inter-connected. They are not entirely the parts of an interesting jigsaw puzzle. True they place personal clues on the individual piece used in the game but they are the result of the writer's conscious attempt over several years to increase the value of each piece by using pictures that illustrate the shape so that it fits more easily with its fellows. The new problem of lung pathogenesis continues to be instructive while it whets the appetite for still further problems.

The ultimate aim of this handbook is to bring added help to the student undergraduate and post-graduate in the assessment of the living and hanging pathogenesis of his individual patient only by so doing an ever increasing benefit that under problem find actual treatment which must be the object and is always the final true reward of the happy physician.

My thanks are due to Sir W. H. Longdon Brown for his kindness in writing the Foreword to F. L. J. A. Kennedy for his assistance in reading proof and in preparing the index and to Mr. J. R. of Messrs. J. & A. Churchill Ltd. for his valuable help in these days of difficulty with paper and print.

R. R. TRAIL

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CHEST EXAMINATION

SECTION I APPLIED ANATOMY

CHAPTER I

THE BRONCHIAL TREE

The trachea lies behind the great vessels embedded in the elastic areolar tissue which envelopes all the structures lying in the mediastinum. It divides at about the level of the fifth dorsal vertebra into its two main branches the right and left bronchi. To this point it can be seen on the normal postero-anterior film of the chest as a clear area, bearing slightly to the right of the mid line of the thorax.

The point of division is important in all conditions which cause displacement of the mediastinum. In that it lies not far below the third dorsal vertebra opposite which is the weakest part of the mediastinum. On this point the mediastinum swings laterally as on a hinge. Any pull on the lung structures connected with one main bronchus is easily transmitted along the lower part of the trachea to this weak point. We shall see later that the trachea responds more than any other mediastinal structure to lung changes, and that therefore if we find evidence of its disturbance we can reason backwards to find the cause in such lung changes. Fortunately its movement is quickly reflected on the sternomastoid muscle so that we have an easily demonstrable sign of its displacement.

The anatomical relation of the muscle explain this reaction (see Fig. 1). On the anterior surface of the trachea lies a band of tissue called the pretracheal fascia connected below with the elastic areolar tissue of the mediastinum, and above with the

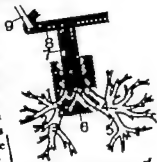


FIG. 1 The Mechanism of the Sternomastoid Sign.

- 1 The right upper lobe bronchus.
- 2 The right middle lobe bronchus.
- 3 The right lower lobe bronchus.
- 4 The left upper lobe bronchus.
- 5 The left lower lobe bronchus.
- 6 The areolar tissue of the mediastinum.
- 7 The pretracheal fascia.
- 8 The fascia of the neck.
- 9 The sternomastoid muscle.



cases of subternal thyroid and in degeneration of one lobe of the gland which pushes the trachea out of its central position.

Along with other mediastinal structures the trachea is displaced in the common abnormality of dorsal scoliosis which is usually convex to the right. This is really a tilting of the thoracic cage pushing the right lower half forwards and outwards and pulling the left lower half inwards and backwards as viewed anteriorly. When we look at the patient from the front we see his right lower ribs forced apart to produce bulging while the left lower half appears to be fallen in. Exactly the opposite is found on looking at the back: the right lower zone is flatter and the left lower zone more prominent than the normal. In the words of the volume of the chest cavity as a whole is not reduced. The effect of this



FIG. 2. Normal postero-anterior film.

distortion on the shadow of the heart and of the main vessels will be discussed in the chapters on the mediastinum; health and disease and on the normal postero-anterior film.

deep fascia of the neck. The latter fascia which meets from both sides in the middle line divides as it goes backward to enclose the sterno-mastoid muscle and is thus in close contact with its tendinous part which has its origin at the anterior superior border of the manubrium sterni. We see therefore how it is that tracheal displacement is reflected to an increased tension of the sterno-mastoid muscle in this tendinous part on the same side as that to which the mediastinum is pulled or pushed by traction on one side or pressure from the other.

As we proceed with our studies we shall find that a pull on the trachea is effected by all processes which interfere with the elastic tissue of the lung. This elastic tissue is continuous throughout the bronchial tree and carried from its smallest division to the elastic surround of the air cells indeed with the finest ultimate arterioles it forms the actual alveolar wall.

All branches of the bronchial tree are also intimately surrounded by a binding of connective tissue which acts as a supporting structure like scaffolding. It is resilient and moves in response to movements of the bronchi as they alter in length and diameter by inspiration and expiration but it is not an integral part of the bronchus like the elastic tissue and so its pathological changes have not the same opportunity to reflect themselves on the mediastinum. Its changes appear to act only secondarily. Its commonest pathological change is in the deposition of fibroblasts which produce peribronchial fibrosis. Increase in depth of tissue leads only to ultimate shrinkage and loss of resilience what seems to happen is that the fibrosis obliterates lobules it was meant to support and causes such a drag on others in its neighbourhood that it destroys their elastic tissue after distending them.

Direct and indirect destruction of elastic tissue occurs quite early in adult pulmonary tuberculosis. Material from diseased lobules enters and then blocks the supplying terminal bronchiole. As air cannot now reach the alveoli empty ones collapse, and full ones organize and both lose their elasticity. The trachea is thus pulled to the diseased side and we find a sharp inner border to the sternomastoid muscle on the same side.

The effect of the mediastinal hinge on tracheal displacement is well shown in disease confined to the upper lobes. It is not unusual to find that the part above the third dorsal vertebra is dragged into a definite bow by localized infraclavicular tuberculosis. In the same way a kink in this region is seen in several

THE BRONCHIAL TREE

5

the upper lobe is supplied by two branches convex outwards that come well down towards the left cardiac border supplying the thin triangular part of the lobe that lies in front of the lower lobe.

If any of these normal markings are altered in distribution or have disappeared we can conclude which part of the bronchial tree has been interfered with by disease and so get a very helpful aid to diagnosis.

Thus on the film of right lower lobe pneumonia we see no shadows of the blood supply to the diseased lobe. If we can follow the markings of the rest of the blood supply to the lung we



FIG. 4



FIG. 5



FIG. 6

shall find them quite normal in their distribution (see Fig. 4). If the disease is right pleural effusion we can find the shadows of congested blood vessels in the compressed lung, internal to the shadow cast by the fluid. There is no obliteration of blood supply (see Fig. 5).

On the film of pleural effusion complicating lower lobe pneumonia we shall make out the shadow of the fluid to the periphery internal to it we see no markings of blood supply in the diseased lobe. The rest of the blood supply is normal in distribution (see Fig. 6).

If we are dealing with a case of collapse of the right lower lobe we shall not see two effects on the shadow of the blood supply. First there are no markings in the area of collapse which is more or less opaque and appears to be continuous with the heart shadow. Second as the lower lobe has collapsed and shrunk the shadows

It is interesting to note that the effect of scoliosis on rib-spaces is no longer seen when pulmonary tuberculosis supervenes. Compensatory emphysema seems to undo it.

British and Continental writers give differing descriptions of the bronchial divisions. The latter describe the branchings as monopodial coming off what they consider to be the primary bronchus in the continuous stem from the bifurcation to the base of the lung. British writers have used the nomenclature of Ewart who described the branchings as dichotomous. This is not exactly true because consecutive divisions often follow on each other too closely to demonstrate dichotomy and the individual divisions differ so much in diameter that three or more may seem to arise at one point. We cannot follow these divisions on the chest film, but we can see the main divisions of the vascular supply and these are so similar and so closely approximated to the main bronchial divisions that they form a practical guide to them. They can be traced on a good postero-anterior film throughout three-quarters of the lung fields from their origins in the hilar regions. There is one point of difference that is important when reading abnormal films: blood vessels continue below the shadow of the diaphragm; bronchial shadows do not unless they are enlarged by disease.

The diagram of the blood supply shows the branches of the pulmonary arteries as they appear on a good postero-anterior film. The right pulmonary artery makes a T-shaped shadow at the hilum. It gives off three branches to the upper lobe, one to the middle lobe, and three to the lower lobe. The left artery gives an elbow shaped shadow at the left hilum, about half an inch higher than the right one on a 15 x 2 inch film. It gives five branches to the upper lobe and four to the lower lobe. It will be noted that much of the left lower lobe supply is hidden by the heart shadow and that the lower part of

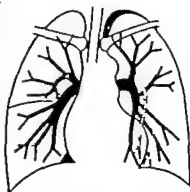


FIG. 3. The Blood Supply. The posterior branch of the right upper lobe blood supply and the blood supply to the lingula of the left upper lobe are shown in dotted lines.

off-shoots because such conditions as tuberculosis, abscess of lung and collapse may be in an area d limited by the air supply from one particular bronchus. This area is cone-shaped th apex of the

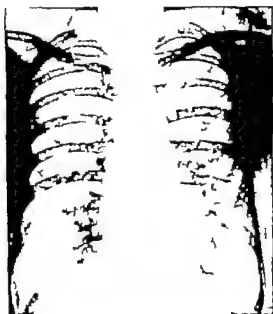


FIG. 9 Congenital cystic disease complicated by collapse and bronchiectasis in the left lower lobe.

come lying t the entrance f the bronchus. This explains why disease of any one such area which lies laterally in th lung will cause triangular loss of transluency on the postero-anterior film.

In 1932 Krause and Glass of America introduced th term broncho-pulmonary segment. They defined it as the area of parenchyma supplied by a constantly placed bronchus wh ch opens off large lobar bronchus and is easily found by bronchoscopy. This was the beginning of proper localization of lung disease. It led to a nomenclature of lung division based on the position of the broncho-pulmonary segment within each lobe as found by the study of films and bronchograms in various positions.

THE BRONCHIAL TREE

has vacated is filled by the upper and mid lobes which enlarge by emphysema so that their blood supply is splayed out as against its normal distribution see Fig (7)



FIG. 7



FIG. 8

Fig 8 shows the effects of collapse of the right upper lobe. There is no sign of the blood supply to the collapsed area that to the middle and lower lobes is spread laterally as against the normal distribution

Now and again we come across subjects in whom the normal division of the bronchi has gone wrong. Development has ceased at one point of bifurcation and instead of a branch we have an air containing space blown out by air from the bronchus and kept open by surrounding lung structures. It may be small or large and is lined by the normal elements of the bronchus. It is known as a congenital cyst of the bronchus. It can be found in forms which maintain their connection with the original bronchus, but many are closed off from the first or later in life. Some contain a serous sterile fluid but most are empty. The walls are thin but well enough defined and the bigger ones may give demonstrable pressure on surrounding structures. When the connective with the original bronchus is maintained through a valvular like flap these cysts may have a clinical significance but the usual form has no effect on the subject. On the film these appear as single or multiple circles sometimes superimposed on each other like beaps of thin curtain rings.

We need not know all the details of the finer bronchial subdivisions but we must know the principal branches and their main

THE BRONCHIAL TREE

respiratory infection the former area will be infected where the patient is lying on his back and the latter where he is lying on his side. Further we shall be aided in treatment as we know the exact location of the abscess we know how to position the patient for postural drainage where to insert the bronchoscope for bronchoscopic drainage or where to operate for surgical drainage.

Similarly we have learnt that bronchiectasis is not necessarily a lobar disease. We now know that the lingular segment only of the left upper lobe is frequently involved that the upper segment of a lower lobe may be entirely clear though its posterior segments are grossly diseased and that only segmental divisions of the main lobar parenchyma may be involved. This is of material importance to the patient and to the thoracic surgeon who can now adapt his technique to deal with disease of less extent than one lobe.

There are four bronchi of particular interest to the student all are in the right lung, three connected with the main upper lobe bronchus and one with the main lower lobe bronchus.

The right upper lobe bronchus has three main divisions the apical the anterior and the posterior. The apical branch goes upwards outwards and a little backwards. It supplies that part of the upper zone of the lung which is weak in supporting structure and so is common site for emphysema and for the first signs of lobar collapse from bronchial blockage. Its air containing cells soon fall in when they are not kept fully supplied by residual air which is the principal method of maintaining their position. They may get stretched and become emphysematous by their lack of support and when they rupture by such stretching may cause a tearing of the visceral pleura to produce spontaneous pneumothorax.

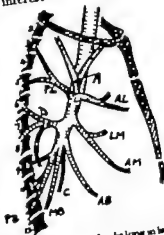


FIG. 11. Branches of right lung in lateral view to show relations of bronches referred to in text.

A, apical; AB, anterior basal; AL, anterior-lateral; LM, lingular middle; AM, anterior middle; C, Cardiac; D, dorsal; LMB, lingular middle; MLB, middle lobe; PM, posterior middle; PBL, posterior-lateral (posterior apical, with dorsal branch).

This type of pneumothorax is known

The actual shape and size of the major segments differ but the pattern of each has individual characters so that each lobe can be divided into recognizable areas. There are nine of these in the right lung and eight in the left lung. The lingular section of the left upper lobe is comparable with the right middle lobe other changes in the pattern of the left lung are due to a combination of two segments in the upper and two in the lower lobe.

Abnormal shadows seen on films which approximate to these known areas and bear similar relations to the bony thorax can therefore be identified as disease conditions in definite segments. It is always necessary however to supplement this opinion with bronchoscopy. One of the great advantages of this method of localization is that a segmental shadow now focuses attention on its supplying bronchus. The shadow may be due to collapse or consolidation the bronchus will almost certainly be found to be abnormal either blocked or constricted and what is of material importance to the patient, the cause may be something removable.

The benefits to the diagnostician are very marked. Thus through the work of Blair we know that the lesions of pneumonia seldom stick to segmental boundaries they either affect an area less than that of a whole segment or they overflow into adjoining segments so that a truly segmental shadow is against the diagnosis of simple pneumonia. It is also against the diagnosis of phthisis except in its bronchial form but truly segmental shadows occur in childhood tuberculosis where bronchus is constricted by an enlarged gland.

We now realize that lung abscess is frequently found in the upper posterior part of the low lobe and the anterior portion of the upper lobe. We see why this can occur from gravity in upper

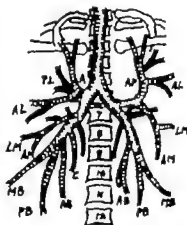


FIG. 10 The anterior view of the bronchial tree, showing the main branches to the lung segments named with them.

A, apical; AB, anterior base; AL, antero-lateral; AM, anterior middle; AP, apico-posterior; C, Cardiac; LM, lateral middle; MB, middle base; PB, posterior base; PL, postero-lateral.

THE BRONCHIAL TREE

respiratory infection the former area will be infected where the patient is lying on his back and the latter where he is lying on his side. Further we shall be aided in treatment as we know the exact location of the abscess where to insert the bronchoscope for postural drainage or where to operate for surgical drainage of bronchoscopic drainage.

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The right upper lobe or primary bronchus has three main divisions the apical the anterior and the posterior. The apical branch goes upwards outwards and little backwards. It supplies that part of the upper zone of the lung which is a common site for emphysema and is the first to collapse in when bronchial blockage is present.

Its containing cells soon fall in when they are not kept fully supplied by residual air which is the principal method of maintaining their position. They may get stretched and become emphysematous by the lack of support and when they rupture by such stretching may cause a ring of thin visceral pleura to produce a spontaneous pneumothorax.

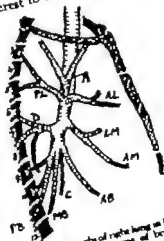


FIG. 11. Branches of right lung in lateral view to show relations of branches referred to in text.

AL, apical; AB, anterior basal; LM, lateral middle; AM, anterior middle; PB, posterior basal; MB, middle basal; PL, posterior lateral; PLB, posterior lateral basal; PLB, posterior lateral basal; PLB, posterior lateral basal.

This type of pneumothorax is known as a visceral pleura to produce a

simple to distinguish it from pneumothorax of tuberculous origin

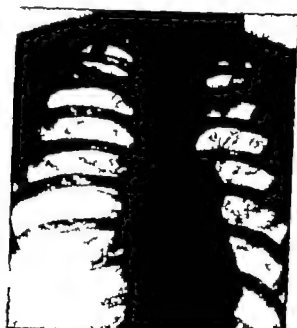


FIG. 12. Simple spontaneous pneumothorax, with emphysematous bullae on the upper border of the collapsed lung

The apical branch is not often affected by pneumonia but is especially important in that it gives off a dorsal branch supplying a sector of the parenchyma commonly affected in adult tuberculosis. When this occurs we see in the supraclavicular area of the lung field small rounded deposits more or less defined in outline and dense in the centre.

Reference will be made later to the difficulties in interpretation of shadows in this area. In the meantime it is sufficient to say that it is a fairly safe rule not to read evidence of apical tuberculosis unless one can see quite definite areas of loss of translucency or at least five rounded dots lying under internal or external to the shadow of the anterior end of the first rib above the collar bone.

The axillary branch comes off the main trunk close to the pectoral branch towards the centre of the upper mid zone of the right lung field and is directed outwards to supply the outer area of the lobe below the clavicular zone.



FIG. 13. Apical tuberculosis of the left lung.

When this branch is involved in such a disease as pneumonia we have therefore on the film a triangular area of loss of transparency which has its apex at the entrance of the branch, and its base along the axilla. Like the apical branch it gives off a dorsal bronchus important in adult tuberculosis.

Later in the section on applied pathology we shall discuss the differential diagnosis of the film shadow of partial pneumonia involving the axillary bronchus and of adult pulmonary tuberculosis at the outer base of the right upper lobe.

The pectoral branch bends a little outward and then comes forward in a semicircular fashion, the accompanying branch of the pulmonary artery sometimes showing up prominently in a completely normal film as if it were half empty with the other half being filled by the mediastinal shadow. If the lung sector of this bronchus is involved in pneumonia there is a triangular loss of transparency rather like the error image of the axillary sector, this time the base is against the mediastinal



FIG. 14. Showing pneumonia of the pectoral and axillary branches of the right upper lobe, and pneumonia of the lower lobe.

shadow and the apex towards the centre of the upper mid zone of the lung. In some cases the shadow is first noted when the apex

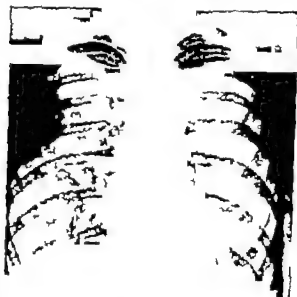


FIG. 15 : Pneumonia of the apical branch of the right upper lobe bronchus.

of the triangle is rounded off this may cause considerable difficulty in diagnosis as against tumour or cyst. Serial films usually clinch the diagnosis by the comparatively rapid changes that occur in the partial pneumonia as the disease resolves.

The principal branch to remember from the right lower lobe bronchus is the posterior horizontal (dorsal or first dorsal branch). It does exactly what its name describes. It comes off the main stem of the lower lobe bronchus posteriorly practically opposite the origin of the middle lobe bronchus and therefore near the centre of the hilar shadow or the film. It goes almost horizontally backwards. If therefore it is involved in disease the abnormal shadow is close to the hilum. This is why so-called central pneumonia commoner in children than in adults appears to be part of the mediastinum. As it hooks backwards over the apex of the lower lobe this branch is a common site for blockage. Its long segment is often involved in adult phthisis. Septic material is easily caught in it to give the foundations for an abscess while the

accompanying lymphatics round its blood supply are equally easily occluded by phagocytes containing tubercle bacilli

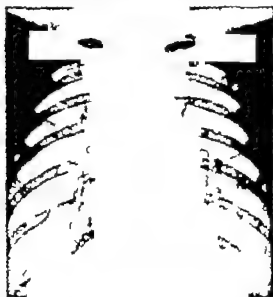


FIG. 16. Perforation of the pectoral branch of right upper lobe bronchus.

Its occlusion is a common cause of characteristic film of adult tuberculosis. When a tubercleoma has formed in this area, close inspection of the postero-anterior film will often reveal a line running downwards and outwards from the hilum and forming the outer boundary of a triangular loss of translucency. The lingula of the lower lobe has shrunk downwards and inward on to the main interlobar fissure. There is collapse of the apex of the lower lobe.



FIG. 17

of the right lower lobe bronchus

They are similarly the earliest sites for evidence of blockage of lymphatics in diseases of dust inhalation such as silicosis. The



FIG. 18. Pulmonary tuberculosis. The collapsing apex of right lower lobe as tongue-shaped shadow in third interspace.

broncho-pulmonary areas supplied by these branches are like all such areas conical in shape but on the postero-anterior film are photographed along a line drawn through their narrowest point at the entry of the bronchiole.



FIG. 19. Common sites of tuberculosis in broncho-pulmonary segments supplied by different bronchi.

It follows therefore that disease in them throw a rounded or oval shadow because the bronchus and also therefore the segment of lung it supplies lie antero-posteriorly in the chest. An antero-posterior film (i.e. one with the patient's back to the screen) will show how the diseased area goes backwards. The shadow is much bigger.

It is important to note that the right middle lobe bronchus comes off the right main bronchus at an angle of almost 90 degrees and that its branches go out almost horizontally towards the periphery of the lung. This makes the sectors of parenchyma supplied by its bronchioles appear as small triangles with their apices inwards and their

THE BRONCHIAL TREE

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bases outwards when they are affected by disease as the trachea is difficult to drain and it also makes any of blockage



FIG. 20. Anatomic focus in left lower lobe.

and we shall find in this the explanation for the comparative frequency of complications in this lobe. Diseases which in other lobes of the lung go on such as emphysema or worry to the physician. We have already noted that all branches of the bronchi carry throughout the lung. By this fact we are able to return to their normal position of rest and they have been elongated and opened up in the act of respiration. If flow of air is stopped by such distribution the lung tissue is anchored to the hilum, so that the lung is also pulled up. It is pulled outwards to return to this point of anchorage.

Similarly all branches carry the tracheal structure of cartilage and muscle and have round them sheaths of connective tissue supporting them but moving with them in respiration. These sheaths carry the blood vessels from the bronchial artery and are loosely related to the accompanying circulation from the pulmonary artery. Through them run also the lymph channels. The end out strands which support the lung parenchyma. We shall consider the connective tissue in more detail when dealing with the structure and function of the alveoli.

CHAPTER II

THE ALVEOLI THEIR STRUCTURE AND FUNCTIONS

WHILE the bronchi continue to divide they retain their component cartilage muscle and elastic fibres into the final subdivisions called the terminal bronchioles. There is much argument among research workers on the details of what exactly happens thereafter until we reach the air cells or alveoli but what matters to the student is that while all the bronchial elements remain up to the terminal bronchiole, the cartilage disappears after this subdivision but strong muscle and elastic fibres continue into the final cylindrical channel the bronchiolus respiratorius. This bronchiole with its communicating air-cells forms that minute section of lung called the acinus.

The Acinus

The acinus is so important in the understanding of many diseases of the chest and is the basis of explanation of so many physical and X ray findings that we must pay some particular attention to its details of construction and its relations both anatomical and physiological.

Its bunches of air cells or alveoli contain the residual air of the lung that is the air that remains after a maximal expiratory effort has been made to expel what is called supplemental air. In other words this residual air can be expelled only by collapse of the cells. It is in contact with cubical epithelium, which has replaced the columnar ciliated epithelium of the bronchi, and through it is brought

into close contact with the finest capillaries of the blood supply for gaseous interchange.

Residual air is at atmospheric pressure being the same as that



FIG. 21 The Acinus.

- 1 Termination of cartilage.
- 2 Termination of muscle fibres.
- 3 Termination of elastic fibres.
- A. Terminal bronchiole.
- B. Bronchiolus respiratorius.
- C. The acinus.

of inspired air and therefore the same as that continually pressing on the outside of the thoracic cage. In other words, it is one offset to the attempt of outside air to push in the bony thorax. The same ultimate effect would be got by any other occupying material of the alveoli, e.g. pneumonic exudate as long as the pressure is the same. Thus we shall see is an important point in the differential diagnosis of chest disease.

The muscle fibres continue along the bronchiolus respiratorius and get steadily stronger until we reach the last passage that can be said to be lumen where we must not they finish by becoming circular only instead of longitudinal and circular. This anatomy explains many fundamentals of normal physiological lung movement and of the changes instituted by disease processes. By the act of inspiration there is a lengthening of the muscle fibres throughout the whole bronchial tree as far as the acinus, thus there is stretching and opening up of every air-containing lumen. The muscle is smooth muscle and so maintains movements along the bronchi even when the lung is at rest thus keeping up the flow of air to the alveoli with an action similar to that of the bagpipe.

Destruction of muscle in the bronchial wall is the forerunner of serious consequence to the acinus, their residual air is not maintained and they are apt to collapse. Muscle spasm in such disease as asthma has also serious effect, the steady spasm induced by the disease even during lung rest is accentuated by the movements of respiration, and is communicated to the regular fibres which lose their like fashion over the entrance to the acinus. The patient has therefore increasing distress in his difficulty in maintaining sufficient residual air. Such an addition to his already established emphysema explains the extreme cyanosis often noted in the bronchial asthma of the chronic bronchitic. This point will be better understood when we consider the effects of emphysema.

Elastic tissue continues all the way to the alveoli finishing round each as if it were net bag round tennis ball. Continues from the main bronchial division in the mediastinum which may be considered as its point of anchorage. It is kept on constant stretch in every direction throughout the lung fields by the air in the alveoli. What this comes to is that the residual air in each alveolus is like a small distensible bladder which has two physiological effects. First it keeps up tension on the elastic tissue so that the pull on the anchorage is constant and therefore the lung is always attempting to retract towards the hilum.

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Second it has a marked effect on the strength of the outward pull of the pressure in the pleural cavity which is below that of the atmosphere and is therefore called the sub-atmospheric or negative intrapleural pressure. The

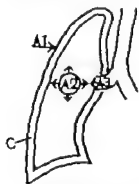


FIG. 22.

- A1 Air pressing on the chest wall.
A2. Air in alveolus.
A3. Inspired air in main bronchus.
C. Inter-pleural space.

maintenance of residual air at a definite level is thus a necessity for proper physiological working of the lung both at rest and during the act of inspiration. If it is cut off by blockage of its supplying channel or destroyed by the breaking down of its alveolar confining walls we shall find definite changes in the position of the lung and the mediastinum. A few examples will help us to understand the ultimate effects of certain disease processes by noting the physiological effect of their pathological changes on lung elasticity.

(a) Destruction of elastic tissue along the bronchial wall occurs in bronchiectasis and tuberculosis. As some of the tension is thus removed from the point of anchorage of the elastic tissue at the main bronchial division in the mediastinum the lung medial to such a break in continuity tends to contract towards the hilum.

(b) Destruction of muscle in the bronchial wall is seen in tuberculosis and bronchiectasis. Air is not maintained in those lobules dependent for supply on the affected branch because the bagpipe action of the smooth muscle has been lost and the end result is the same as in (a).

(c) Cutting off residual air which maintains the stretch on the elastic surround of the alveoli happens if anything blocks the supplying lumen—pus, blood or a foreign body. This happens in adult phthisis. Tuberculous material from diseased alveoli enters and later blocks the bronchiole supplying them. Empty air cells then collapse, those that still contain material organize and all lose their outward pull on the mediastinum.

The acini are bound together into lobules by connective tissue septa. This connective tissue which runs throughout the lung as a sheath accompanying the bronchial distribution acts as the supporting structure. It binds bundles of air-cells together into lobules and acts as a barrier which tries to confine disease to

these small sectors of lung substance. This function it can perform because it is rich in blood supply which increases markedly in response to irritation thus giving inflammation as the evidence of resistance. Continued inflammation however brings much increase in cells which are later permeated by new capillaries from the swollen blood vessels. The result is the formation of fibroblastic cells which obliterate the new capillaries so that nature's attempt at repair of the damage leads to interstitial fibrosis. Such repair tissue is fraught with danger to the bronchi and the parenchyma. What was formerly a resilient supporting structure is now a fibroid and restricting band which impedes the normal expansion and elongation of the bronchi with inspiration. It also interferes with the blood supply of the bronchial artery which runs through it. Further by its shrinkage it pulls on the surrounding lobules through their supporting septa with which it is connected. In its advanced stages such as are seen in bronchiectasis this traction can actually obliterate alveoli by crushing them.

This combined action of constriction of the bronchi and destruction, and consequent collapse of near by lobules is well demonstrated in the film of early bronchiectasis. We can see that the bronchi of the inner bundle of the right lower lobe are crowded together just outside the lower third of the right cardiac border. Their outlines are congested and enlarged so that they are much more prominent than normal and we know there has been



FIG. 21. Early bronchiectasis. Crowding of bronchi in right cardiophrenic angle.

destruction of the lobules in their immediate neighbourhood because we find there is loss of lung markings in the outer lower zone of the lung where compensatory emphysema has resulted.

20 THE ALVEOLI STRUCTURE AND FUNCTIONS

Later we shall learn that increase in connective tissue round the bronchi is one cause of the increase in the normal striations on the film, and that it gives characteristic dry crepitations when we listen by stethoscope

The Supporting Structure of the Lobule

The connecting septa round lobules can be so distended that it breaks down in the disease of emphysema. This can be recognized on the film as a loss of normal markings. To this point we shall return more fully later in our studies on applied pathology. It can also be broken down by septic material in its blood vessels when this is carried to them as emboli from an infected focus. In this way many small scattered abscesses can appear throughout the connective tissue over large areas of the lung in the disease of pyæmia.

Lymphatic Supply

Lymphatics are in abundant supply in the connective tissue round the lobules. Their presence explains the first steps in the pathogenesis of certain lung diseases. The whole system in the lung is divided into two sets, a superficial and a deep, which communicate with each other in the pleura and at the hilum. The superficial set lies in the pleura, which we shall consider later. The deep set runs with every subdivision of the bronchi, the pulmonary artery and the pulmonary veins, and so it follows that the connective tissue between the lobules has an exceedingly rich lymphatic supply. This connective tissue supply is finally filled with lymph spaces that bathe individual acini, and of this fact we must make especial note, as it is by this route that tubercle bacilli finally enter the lung parenchyma in adult phthisis. The bacilli circulate and multiply in the lymph spaces from which they penetrate the walls of the air-cells.

All lymphoid tissue in the body is meant to be a protecting factor, both at the point of any disease process where it acts as an absorbent agent, and also in its canal system whereby actual body irritants and their products may be carried to the glands. In other words, the glands are the drains, the lymph channels are the gutters. We see these lymph channels act as a conveyor system in silicosis; they transport dust-containing cells from the alveoli to the lymph spaces round the acini, from there to the

connective tissue lymphatics and thence by peri-vascular and peri-bronchial channels to the lymph glands. Lymphoid tissue carries out the same function in childhood tuberculosis draining the primary focus to the glands in the hilum. But in adult tuberculosis of the proliferative type we see that it is the lymph spaces between the alveoli that start the first reaction towards the production of the actinar lesion not only do the lymph spaces fail to perform their proper function in this case but they actually break down to become culture ground for the invading bacilli. All lung lymphoid tissue increases with age. It is the breeding ground for that widespread form of tubercle known as senile phthisis. But it seems to do its utmost to reassert its protective powers. The patient is so little upset constitutionally that he may be a dangerous but unsuspected carrier of the disease and is often treated for what he appears to be a chronic bronchitic.

The Blood Supply

The alveoli are very rich in blood supply the final capillaries of the pulmonary artery being so numerous as to be a component of their walls while their connecting and binding septa of connective tissue have an almost equally abundant supply of nutritive vessels derived from the bronchial arteries. The distribution of these arteries explains many points of difficulty in chest diseases and helps us to understand many of the markings on normal and abnormal klagrams.

The function of the pulmonary artery is oxygenation of the blood stream. It originates from the right ventricle and under the arch of the aorta divides into its right and left branches. At the hilum the right branch lies in front of the right main bronchus the left branch just below the left main bronchus. It follows therefore that the main pulmonary artery and its branches enter in great part into the composition of the hilar shadow on the film. In the lung each artery follows the bronchus lying behind and slightly lateral to them in all their sub-divisions. They are the guide as we noted in the last chapter to the bronchial distribution. We must remember however that while close to the hilum the artery and the bronchus are practically the same size the arteries lessen in size very fast as compared with the bronchus and give off many more branches within the same distance. This is why when looking at an abnormal film we cannot tell whether the radial lines of translucency we see in streaks are blood vessels or bronchi.

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Within a distance of an inch towards the middle of the lung fields a bronchus might be seen as continuous parallel lines with no branches while an artery will not so appear. By the time the pulmonary arterial sub-division has reached an acinus it has only about one fifth of the diameter of the supplying bronchiole to the alveolus.

We must remember however that the arteries can be materially changed in size by various diseases. Thus in congestive failure they may be considerably swollen they then give increased soft markings generalized throughout the lung fields and lessening towards the peripheries of the film. The opposite effect can be noted in conditions associated with extreme anaemia where their thin flaccid tubes are indistinguishable from the normal striations of connective tissue. These changes will of course be accompanied by relative changes in the shape and size of the cardiac shadow and be apparent in both lung fields.

The function of the bronchial artery is to bring necessary nutrition to the framework of the lung. That for the right lung comes usually from the thoracic aorta but occasionally from the first or third intercostal artery the left supply has usually two origins from the thoracic aorta. It is important to remember this difference of origin of the pulmonary and bronchial supplies the latter as direct from the main arterial vessel has the higher pressure so a wound or other cause of a break in its wall can prove much more rapidly fatal than the involvement of an arteriole from the pulmonary artery.

As soon as the bronchial arteries enter the lung they enter the connective tissue layer round the bronchi two or three subdivisions accompanying each bronchial branching. This is why any condition increasing blood supply has the possibilities of marked effects on the bronchial walls why such conditions of long-standing inflammation as chronic bronchitis and bronchiectasis can cause the havoc of fibrosis round the bronchi affected in the disease. Here we have the explanation for the film of chronic bronchitis. The normal supply from the bronchial artery is very rich. When this supply is swollen by chronic inflammation, and brings the consequent deposition of fibroblasts we get the evidence on the film in the appearance of parallel lines in the affected area outlining the larger bronchi in the lower zones of the lung fields. We can find the same effect in other parts of the lung in other diseases—

g In the upper zones in chronic pulmonary tuberculosis

Such rich blood supply is the reason why quite severe hæmorrhage can occur by even small abrasions of the bronchial wall by irritant material inspired into the smallest divisions of the bronchial tree; and why the breaking down of the bronchial wall in bronchiectasis can produce the biggest of all hæmorrhages in lung diseases.

CHAPTER III

THE PLEURA AND THE DIAPHRAGM

THE pleura is in two layers connecting at the hilum. The first or visceral layer is closely applied to the lung. It reflects at the hilum to form the second or parietal layer which lines the inner surfaces of the structures internal to the thoracic cage, and the diaphragm. The parietal layer is sensitive. Pain and severe shock can occur when a needle is pushed through it in the induction of artificial pneumothorax, and the pull of adhesions on its inflamed surface may well be the reason for the acute stabbing pain with each respiration in lobar pneumonia.

The two layers have between them a potential space. They do not adhere in health but move easily on each other by virtue of a thin lining of serous fluid in which the visceral layer can slide on the parietal. In the space there is a pressure which is negative in that it is below that of the atmosphere. A needle inserted in this space and connected with a water manometer shows that the pressure varies on an average between -10 cm. of water pressure with inspiration and -5 cm. with expiration. It therefore exerts a constant outward pull on the elastic tissue of the lung, adding to that of the residual air in the alveoli. Each inspiration adds to the pull thus lengthening and opening up the bronchi to receive the inhaled air.

It follows that any pathological alteration of the negative intrapleural pressure has a marked effect on the elastic tissue of the lung. If it be reduced to the level of atmospheric pressure by air in pneumothorax or fluid in pleural effusion the lung must tend to recede to the anchorage of its elastic tissue at the hilum, retracting to a size less than that of complete expiration. The stretch on the elastic tissue of the lung is now maintained only by the residual alveolar air. Raising of the pressure above that of the atmosphere and therefore above that of residual air will bring pressure collapse of the alveoli and consequent still further retraction of the lung for now the pull of the alveoli on the bronchial elastic tissue is being lessened. As we shall see later when discussing the mediastinum these are the ruling factors in the displacement of mediastinal structures in pleurisy with effusion.

THE PLEURA AND THE DIAPHRAGM

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In going round the lung as described the visceral pleura dips between the lobes lining the under surface of the one above and the upper surface of that below it. One such lining may appear on a normal postero-anterior film of the chest. It is the lesser fissure between the upper and middle lobes of the right lung, and it shows as a thin hair-like line running out almost horizontally from the hilum in the third or fourth rib-interspace. Sometimes it casts a double line as the X rays have cut across it tangentially. Its position is easily recognized when it is forced apart by fluid or its rich lymphatic supply is inflamed or thickened in such diseases as pneumonia, lung abscess and tuberculosis. If it is seen on the postero-anterior film its location and direction should be noted carefully as this may give an indication of disease process that can be clarified only by further film examination—e.g. lateral film. Thus it is displaced upwards and towards the infra-clavicular zone in collapse of the right upper lobe.

Other fissures do not show on the normal postero-anterior film and it may take lateral or lordotic films to show their involvement in pathological conditions.

Some students find difficulty in the interpretation of abnormal films because they forget that the interlobar fissure as they visualize them are but the points of reflection of the pleura which in the normal lung being known as the lingula, ring any lobe must necessarily cover by the shadow cast by normal lung structure from front to back on the postero-anterior film. It follows that density of shadow in developed lobar pneumonia arises by depth of those and not by intensity of disease which is equal in the upper lobe pneumonia to be as dense in its lower limit as in its upper because the lobe is completely encased in it going downwards.

It cannot expect the shadow of right middle lobe pneumonia to be as dense in its upper limit as in its lower because the lobe is not completely encased in it going downwards. It has a dense shadow in the right mid zone which becomes as dilute in the base so that though its lower limit is seen the right diaphragm



FIG. 4. Right upper lobe pneumonia.

This explains too why collapsing lobes cast characteristic shadows why the lesser interlobar fissure throws a heavy density



Fig. 25 Pneumonia of the right middle lobe, and of the left lower lobe.

upwards and outwards as the lingula recedes on to it in right upper lobe collapse. Likewise we realize that when a right lower lobe collapses the lingula has already shrunk downwards a long way before we see on the film the upper limit of the lobe defined by a line running downwards and outwards towards the costo-phrenic angle from a point at the upper edge of the right auricle on the right side of the cardiac shadow (see Fig. 86).

The position of these fissures in the lateral film is discussed in Chapter V.

With such changes in shape of lobes we shall learn to correlate changes in the normal striations that appear on the film by shadows of vascular supply and supporting structure. Thus we have already noted that in right upper lobe collapse we can no longer see the three normal branchings of the pulmonary artery which supply it, while in the rest of the lung field we shall find spread out much more horizontally than normal the blood vessels of the middle and lower lobes which have been blown out by complementary emphysema to occupy the space vacated by the shrunk upper lobe. In this emphysematous area we shall at the same time find much increased translucency as against the normal because the supporting structure of the vesicles is considerably stretched. We therefore make our diagnosis by noting two opposite processes: increased density and increased translucency; one with loss of blood supply the other with spread of blood supply and of supporting structure. In other words where we suspect a change in one type of lung striation we must look for a corresponding change in the other and if we fail to find one in the presence of another on a postero-anterior film we must make other investigations such as the taking of lateral films or the injection of lipiodol. Thus in complete collapse of the left lower lobe we may see on the postero-anterior film only the gross emphysema of the upper lobe filling the

hemithorax we may not be seeing the collapsed lobe because it is completely hidden by the cardiac shadow. Films taken in the postero-anterior and lateral positions before and after the injection of Iotodol will aid in the diagnosis.

The Diaphragm

The diaphragm is the most important muscle in respiration and therefore next to the heart the most important muscle for the maintenance of life. It consists of thin, movable partition lying between the thorax and the abdominal contents. It is convex upwards its right half being slightly higher than its left and taking a position just below the nipple at respiratory rest. There is a central tendinous very strong portion and three fleshy portions which arise from the ribs, the sternum and the spine and are therefore named costal, sternal and vertebral. Of these we must note especially the costal; its origins are from the inner surfaces of the lower six costal cartilages on each side. When the diaphragm is depressed they may be seen on a postero-anterior film in the diaphragmatic insertions as depressions between convexities thus forming a series of waves and such a picture while commoner in emphysema does not necessarily connote any disease process.

They are sometimes wrongly interpreted as evidence of adhesions of old pleurisy. If there is really adhesion of the diaphragm there will be no clear outline but a merging of lung shadows into the deeper density of the muscle.

These normal alterations in the dome may also be confused with the very common finding of tent-like shadow that sits on the diaphragm about its middle third. This is the end result of pleural reaction to the main fissure.

The central tendinous portion may appear as a hump on the middle of the diaphragm. It is without clinical significance but may require investigation against the comparatively rare pleural growth that can develop at this point.

By the contraction of its fleshy portions through the action of the phrenic nerve the diaphragm becomes less convex and so increases the vertical diameter of the thorax in inspiration. In ordinary breathing this range of movement is about three-quarters



FIG. 26. The costal insertions of the diaphragm.

This explains too why collapsing lobes cast characteristic shadows why the lesser interlobar fissure throws a heavy density

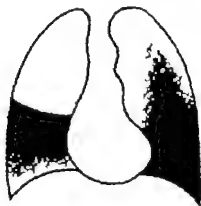


Fig. 25. Pneumonia of the right middle lobe and of the left lower lobe.

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These normal variations in the dome may also be confused with the very common finding of a tent-like shadow that its on the diaphragm about its middle third. This is the end result of a pleural reaction in the main fissure.

The central tendinous portion may appear as a hump on the middle of the diaphragm. It is without clinical significance but may require investigation against the comparatively rare pleural growth that can develop at this point.

By the contraction of its fleshy portions through the action of the phrenic nerve the diaphragm becomes less convex and so increases the vertical diameter of the thorax in inspiration. In ordinary breathing this range of movement is about three-quarters



FIG. 26 The costal insertion of the diaphragm.

of an inch and in every examination of the chest it should be looked for by percussion of the lung bases posteriorly and in the axillae since deep inspiration should move the diaphragm downwards by at least a finger's breadth as shown by the higher pitch of descending air-containing lung.

It follows that interference with the phrenic nerve at any point throughout its course over the cupola and past the hilum to the insertion of its fibres will cause rise of the diaphragm. Evidence of this is common nowadays in the films of patients in whom the nerve has been crushed or evulsed as a treatment for pulmonary tuberculosis. Such films demonstrate that the diaphragm as it moves downwards in inspiration exerts a distinct pull on the apex of the lung, for when it is immobilized an apical tuberculous lesion shrinks and gets the rest it requires to aid healing.

Adhesion between its pleura lining and the visceral pleura of the lobes in contact with it will fix the diaphragm while emphysema will depress and flatten it. Contraction of the lung by loss of elasticity will cause it to rise *e.g.* in collapse of the right middle lobe. The recognition of such changes in its position and movement by physical and film examination can therefore be of very distinct help in the diagnosis of underlying lung disease.

CHAPTER IV LUNG AND MEDIASTINAL MOVEMENT IN HEALTH AND DISEASE

THE mediastinum is a space running antero-posteriorly to the middle line of the thorax between the sternum and the spine. It shows on the postero-anterior film as the shadow of parts of its contents so that we can identify the trachea to a point just above its bifurcation, the outline of the cardiac shadow, the aortic notch, and the structures that make up each hilum. As we have already noted, all these mediastinal structures are embedded in areolar tissue which covers and binds them together but yet is elastic in composition, so that it can move with systole and diastole of the heart and the great blood vessels.

We have already noted too that it is connected by the layer of pretracheal fascia with the investing fascia of the structures in the neck and that thereby its displacement to one side or the other of its central position is reflected to an increased tension of the tendinous portion of the sternomastoid muscle on the same side.

Again we saw that the mediastinum has a weak point on which it swings as on a hinge and that the trachea can move laterally both above and below this hinge. For example, that it can be definitely linked by disease localized in the parenchyma below the collar bone. The heart is displaced much less readily. A much heavier structure it has its greatest weight in the lower mediastinum so that it is moved laterally only by disease conditions in the lower half of the thorax or throughout one whole side of the thorax. Thus the apex beat is seldom abnormal in position in tuberculous of one upper lobe but it can be moved quite markedly to the opposite side in the presence of pleural effusion and to the same side in post-pneumonic fibrosis, this being disease which commonly affects the whole of one lung field.

An important point to note is that the heart will move laterally. Its long axis much more readily than it will move quite markedly explains the anomalous outline it presents in the normal abnormality of scoliosis which has its usual concavity to the left. In lying into this hollow the heart swings on its long axis to its right border.

receding and its left border coming forward so bringing the pulmonary conus on the left side into undue prominence and



FIG. 27 Marked scoliosis with convexity to the right.

uncovering the right hilar region. On the postero-anterior film there is therefore a filling in of the normal triangle of translucency that has its apex between the aortic notch and the normal prominence of the conus giving an outline that simulates that of mitral stenosis on the left side while the shadows of the right hilum and of the branches of the pulmonary artery to the right lower lobe stand out prominently. Such abnormalities might be read as evidence of disease if we did not note the accompanying widening of the right lower rib-interspaces and the compensating fall-in of the left lower rib-interspaces. We can see too why it is by this tendency to swing on its long axis that the position of the heart at respiratory rest may mislead us in such a disease as collapse of the right lower lobe. As it shrinks from such a cause as bronchial carcinoma the lobe goes downwards inwards and backwards and causes the heart to swing on its long axis so that the right lower

border recedes towards the spine the left lower border comes forward. Let the whole heart go backwards. If we have to rely on physical signs for our diagnosis we shall often find more help towards the opinion that the mediastinum is affected by noting the tense right sterno-mastoid than by concluding anything from the position of the apex beat. If we can watch the patient on the screen however we shall see the heart move laterally and bodily to the right in its lower half with each inspiration and come back to its former position with expiration. This is because the intra pleural pressure becomes more negative at each inspiration and draws the whole mediastinum towards the periphery.

A little later we shall see why the heart goes backwards in collapse of the right or left lower lobe.

Experience shows that mobility of mediastinal structures varies considerably in different individuals. The pressure in the pleural cavity in two subjects may be the same as judged by the same manometer in conditions as nearly equal as we can make them yet the amount of displacement from normal in disease of seemingly equal extent may be entirely different. In one small pneumothorax can cause great bulging of the anterior mediastinum in another it will hardly move this area. This means that the mediastinum is never a fixed and invariable entity. We cannot by working backwards get an infallible estimate of the amount of lung disease by having it on the amount of mediastinal shift and all we can say is that some damage has taken place. On the other hand we must realize that disease of the mediastinum can have definite stiffening effect thickening of the mediastinal pleura can combat peripheral changes of pressure. Thus we may get much more falling in of ribs and raising of the diaphragm than we would expect from the amount of damage evident in the parenchyma.

The mediastinum must in any be envisaged as one entity not as something in two halves one for each hemi-thorax. It is one central basic structure both maintaining and being maintained by one intra-thoracic pressure. If there is a fall of pressure on one side of the thorax there is an immediate emphysema on the left other. Fibrosis on the right implies emphysema on the left. Now we can understand why the heart goes backwards in right lower lobe collapse. The pressure in the right lower mediastinum is lessened. It cannot put up normal resistance to the pressure in the left lower mediastinum with the result that the left lower

lobe moves across the front of the heart and pushes it backwards.

We are now in a position to sum up our considerations of applied anatomy to discuss the action of the various lung and intra pleural forces on the mediastinum in health and see how they react on each other and on this central elastic structure in disease.



FIG 28 The forces acting on the mediastinum.

A1 Air pressure on chest wall.

A2 Air in alveoli.

A3 Air inspired into bronchus.

B Elastic tissue.

C Intra-pleural pressure.

D Mediastinum.

In the accompanying diagram, D represents the mediastinum. A represents atmospheric pressure which acts in three ways —

- (1) As A_1 pressing in on the chest wall
- (2) As A_2 the pressure of residual air

In the alveoli we have seen that this is maintained by the bag-pipe action of the smooth muscles of the bronchi. A_2 acts in every direction throughout the lung fields pressing against the periphery the diaphragm and the mediastinum. We have seen it demonstrate its force against the mediastinum in collapse of the right lower lobe. It also presses on surrounding alveoli so if it is lost in any area the surrounding alveoli immediately distend, giving localized emphysema.

- (3) As A_3 the air entering by the main bronchus

B represents the elastic tissue of the lung, which runs with every bronchial division from the hilum to the alveoli. The more a piece of elastic is stretched the more it tries to contract to its fixed point. As lung elastic tissue is on constant stretch by the action of the residual air in the alveoli (A_2) it is always attempting to pull the lung towards to its anchorage at the hilum, where it is continuous with the elastic tissue of the trachea. Therefore in proportion with the destruction of the elastic tissue the lung will always contract towards the hilum.

C represents the sub-atmospheric negative pressure in the pleural cavity. This exerts a steady outward pull on the lung, and is increased with each inspiration.

Again we must remind ourselves that all these forces are acting in exactly the opposite direction against the mediastinum on the

LUNG AND MEDIASTINAL MOVEMENT 33

opposite side A_2 on the right is pushing to the left A_2 on the left is pushing to the right. Thus the central balanced position of the mediastinal structures.

Let us now consider examples of disease conditions affecting each of these factors and see how they react on the mediastinum.

A is constant factor in all ordinary conditions of life in health and disease.

A may alter by such conditions as —

(1) blockage of the bronchus through carcinoma or septic r caseated material.

(2) destruction of the alveoli as in tuberculosis of adult type.

There is therefore loss of residual air. A_1 has less resistance offered to it so the hemi thorax tends to shrink and the ribs tend to fall in. There is less resistance to the diaphragm so it will rise higher at rest and with complete expiration. There is less resistance to neighbouring lobes and so they show emphysema. There is less resistance to the mediastinum so the force on the opposite side will push the mediastinum to the diseased side. Finally there is less pull on the elastic tissue anchored to the trachea through the main bronchus and so the lung will retract and drag the whole lung outwards. We saw an extreme example of this action of C in the detailed discussion of what happens in collapse of the right lower lobe. We noted that the lower half of the mediastinum including the lower half of the heart, wings out to the right with each inspiration, and comes back to more central position with each expiration.

We can represent this in a simple line drawing (see Fig. 28) remembering that all processes are acting equally in the reverse direction in the opposite hemi-thorax.

A is destroyed. B therefore shrinks towards D and C now unopposed pulls B and D outwards. The mediastinum therefore drifts to the side of the atelectasis, the amount depending on how much of A_2 is destroyed. We see now why it is that we get positive sternomastoid sign with loss of residual air. It is not due directly to the collapsed tissue but to such lung tissue as remains unaffected and is dragged over bodily by the intra-pleural pressure. Its drag is communicated to the trachea.

If we have followed these arguments we can see why such diseases as unilateral tuberculosis or bronchiectasis must displace the mediastinum to the side of the lesion.

We have here too an explanation of cavity formation. There is marked localized loss of elastic tissue by organization and collapse of lobules and therefore loss of the pressure of their residual air. Immediately surrounding lobules are unopposed in their own inherent pressure and blow out to become emphysematous and so their weakened walls are dragged in every direction towards the periphery both by those external to them and by the intra-pleural pressure. Apart altogether from any extension of the actual disease process there is physical force behind the production of increasing cavitation.

It is possible to follow now why diseases which replace residual air by material which keeps the alveolar walls distended cause no displacement of the mediastinum unless they overfill the vesicles and so pull the mediastinum a little towards them. Pneumonia fills the alveoli with exudate they cannot move but they keep the elastic tissue of the lung on stretch as full inspiration does. Therefore uncomplicated lobar pneumonia does not give a sternomastoid sign nor does an uncomplicated abscess of lung which as we shall discuss later is merely a filling of lobules by inspired septal material. Tuberculosis on the other hand will do so. It destroys the alveoli. Collapse of the lung does. It takes away residual air. We have therefore here very distinct aid towards differential diagnosis.

Let us now consider disease conditions affecting C the sub-atmospheric negative intra-pleural pressure. If we have a case of adhesive pleurisy C disappears. A now attempts to fill the space vacated by C and drags B and therefore D with it. We get a positive sternomastoid sign on the same side as the disease.

If fluid or air enters the pleural space to remove the negative pressure and bring it to zero A_2 alone maintains stretch on B the lung retracts to the hilum and is not moved outwards by inspiration. But the force in the opposite pleural cavity is correspondingly greater and so we have a drag to this opposite side on which the sternomastoid sign will be positive. In other words it does not require actual push from the right. In a right-sided pleural effusion, to produce mediastinal displacement to the left.

If the intra-pleural pressure rises above atmospheric by increasing air or fluid it will be greater than A_2 . The lung is now pushed in by pressure collapse. The outward pull of A_2 is lost to B which retracts still more towards D. Thus fluid in the right pleural cavity pushes on the mediastinum and at the same time

the pull from the left is still more strengthened and we have now very marked left transverse tension.

Much will depend now on the condition of B. If there is much fibrosis of lung opposing the elasticity of B the lung will not retract to the mediastinum D but only move towards D if it is pushed on bodily. Thus a pneumothorax which complicates established pulmonary tuberculosis can give much more displacement of the mediastinum than simple non-tuberculous pneumothorax which is due to rupture of bulla. The lung without disease collapses quite readily.

If we follow this explanation of lung and mediastinal movement we shall be in a better position to understand the physical and X-ray signs of those commoner chest diseases whose pathological changes are considered in Section IV.

CHAPTER V

THE NORMAL CHEST FILM

A FULL understanding of what constitutes a normal film of the chest is a necessity for the student who hopes to interpret the abnormal. The following description relates only to the points on a postero-anterior and a lateral film which concern the average practising physician. It does not go into all the finer detail which concerns the specialist chest physician, the cardiologist or the pure radiologist, which detail the student may find in specialized text books. It tries to set forth only those points which the physician can correlate with his physical signs and their causative pathology so that he can go beyond the merely objective reading of abnormal lung shadows.

Some preliminary observations on prime principles must be set out. Thus we have already noted that a film is merely a composite photograph of superimposed shadows of various structures through which the rays pass. With shadows of disease at any one depth we shall therefore see the shadows of normal structures in front of or behind them. An early adult tuberculous focus is completely homogeneous to tomograph examination and much more defined on an antero-posterior than on a postero-anterior film. On this last film it may appear to be striated because being a posterior lesion it is shown with much normal lung in front of it, and such normal lung will now throw its striations in greater comparative relief against the background of the disease focus. In the same way we shall see varying shadows of a bronchopulmonary segment in the lung fields. If it be supplied by a laterally directed bronchiole this cone of tissue will appear on the postero-anterior film as a triangle with its apex at the point of entry of the bronchiole. If it be supplied by a bronchiole that goes dorsally it will appear circular or oval as photographed along a line between its apex and the centre of its circular base.

A good film is the result of good positioning, proper exposure as judged by muscular development of the subject and good processing. Although the result is by no means a stereoscopic picture there is no doubt in the mind of the writer that viewing such a film first at close quarters and then at a distance of some

5 feet will tell the observer much of the composition of normal shadows. If not of their depth in the lung fields.

Of late years medical literature has shown much less variation in the objective and interpretative readings of chest disease because certain criteria of a good postero-anterior film have been more or less generally accepted. These are that it may show the lesser fissure of the right lung in the third or fourth interspace; that it should show the superior vena cava with density slightly less than that of the heart, and that it must show the shadows of the branches of the pulmonary arteries radiating from the hilum throughout some three-quarters of the lung fields.

It must be borne in mind that the hilar shadows are themselves predominantly vascular. As the pulmonary arteries and veins give the main part of the rather indefinite semi-circular opacity on each side of the cardiac shadow it follows that this opacity will be increased by all conditions characterized by hyperemia. Seeing the left one is already denser by the prominence of the conus in the normal abnormality of scoliosis, such a condition as broncho-pneumonia in a scollitic subject will produce very heavy left hilar density both during the acute stage of the disease and for long after clinical recovery.

The Normal Postero-anterior Film

The first lesson the student must learn is to read every film normal and abnormal on a definite pre-arranged plan. If this is not done consistently even the expert observer may find his eye has been arrested by some striking area of density or contrast or by one interesting detail in one area of the lung fields and that he has missed an abnormality of lesser extent or contrast that is nevertheless of the first importance in diagnosis. For example a filling in of one costo-phrenic angle is dependent for the interpretation of its real significance on the presence or absence of a lung focus. We have already noted how a grave mistake may be made in the interpretation of the film of a simple scoliosis if we do not connect the variations in the heart outline and the right hilar shadows with the changes in size of the lower rib interspaces.

A simple plan is to read every postero-anterior film under the following seven headings —

- (1) The thoracic cage
- (2) The trachea; shape and position

- (3) The heart size shape and position
- (4) The diaphragm position and contour
- (5) The upper zones of the lung fields
- (6) The middle zones
- (7) The lower zones comparing the right with the left in each case

The Thoracic Cage

The first thing the student of chest films should learn is to recognize the shadows of the bony thorax and those of muscle fascia and skin that can appear on a normal film

The ribs are seen more clearly or less clearly than normal in certain pleural and lung diseases. Tuberculous pleural effusion displaces the lung shadows inwards through the even light loss of translucency cast by the fluid in the periphery the ribs show up more clearly than normal. They are still more prominent in the film of pneumothorax. In empyema and pneumonia they are less prominent owing to the dense shadows thrown by these diseases.

The anterior ends of the ribs often continue into the shadow of calcification of cartilage which is more dense and less even, so



FIG. 29 Fusion of anterior ends of first and second right ribs.



FIG. 30 On the right an under-developed first rib. On the left half- and socket junction of first and second ribs.

that it can simulate the shadow cast by pulmonary disease especially tuberculosis. Usually the condition is seen in more than one rib though it is commonest in the first rib. Even then it is almost always bilateral and it is a fairly safe aid to remember that lung lesions are rarely collar position and characteristics on both sides of the chest.

Fusion and bifurcation of the anterior ends of ribs is common. Special films may be necessary to discriminate between the shadows and those of underlying lung disease. The commonest of all these normal abnormalities is fusion of the anterior end of the first and second ribs.

Points of intersection and overlap of rib-shadow can cause considerable difficulty. This is especially so at the apices where the shadow of the blade of the sternomastoid muscle and skin and fascial folds complicate



FIG. 31 It shows how tuberculous foci may be hidden by rib shadows.

the picture. Fig. 32 illustrates how these shadows may be recognized. That cast by the sterno-mastoid muscle may need

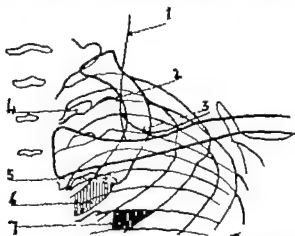


FIG. 32. Drawing to show normal shadows at the apex which may cause difficulty in reading films. 1 = Sternomastoid muscle. 2 = Costal tubercle of third rib. 3 = Shadow due to skin and fascia. 4 = Transverse process of vertebra. 5 = Tip of first rib. 6 = Calcification of first costal cartilage. 7 = Area of density due to superimposition of the shadows of two ribs crossing one another. Note also the expansion shown on the course of the second rib as it crosses the clavicle.

careful scrutiny as it lies in the area involved in tuberculosis arising in and around the posterior apical branch of the upper lobe bronchus.

Bad positioning causes the shadows of the scapulae to obscure the outer parts of the lung fields but these should be recognized as readily as the rounded shadows due to the breasts in the lower zones in females. The shadows of the pectoral muscles and their blood supply may give quite pronounced striations and loss of translucency in the infra-clavicular and mid zones of the lung fields especially on the right side but generally in this case the lower edge of the pectorals can be seen as a sharp line running downwards and inwards.

The Trachea

The trachea should be seen as a band in front of the vertebrae slightly inclined to the right and with no clearly defined walls. It may be a little to one side in scoliosis and is generally seen as far as its bifurcation at the level of the fifth dorsal vertebra.

The Heart

An understanding of the normal cardiac outline is a necessity, for it alteration accompanies and can aid in the diagnosis of diseases which are primarily pulmonary in origin. The following description does not give details required for the study of primary cardiac disease.

The cardiac outline shows as an opaque pear-shaped shadow. The transverse diameter is of no pathological significance unless the heart be of the longitudinal type; it cannot help where it is of the squat transverse type. In this latter case the outline of the left lower border is usually indefinite, as the heart is pushed upwards by the diaphragm. The student should learn to recognize that the shadow of such a heart lessens steadily from its mid-point till it reaches the inferior border; he can still see the density of the diaphragm all the way to the cardiophrenic angle. He will then have no fear of confusing it with the characteristic shadow of lower lobe collapse (compare Figs 86 and 87). Here there will be dense loss of translucency filling the cardiophrenic angle; the opacity is equal to that of the mid-point of the cardiac shadow with which it is continuous. Its outer border is sharply defined. The diaphragm does not show as definite lines; outline angle is abnormally free from emphysematous upper lobe

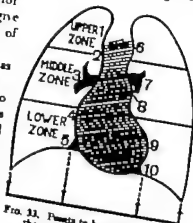


FIG. 33. Points to be noted in reading the postero-anterior film of the chest.



FIG. 34. Chronic bronchitis and emphysema showing a squat heart, an enlarged aorta, splashing of the ribs and flattening of the diaphragm.

below it, while the costo-phrenic angle is abnormally free from emphysematous upper lobe

Two measurements are taken in assessment of the size of the longitudinal heart —

- (1) The transverse diameter of the heart that is the distance between two parallel perpendiculars drawn downwards from the outermost parts of its shadow
- (2) The internal diameter of the chest that is the distance between the outermost points of the thorax along a line drawn horizontally just over the right diaphragm

For normality (1) should be just under half (2)

Let us now follow the cardiac outline starting at the right upper border and working downwards and following the same order on the left side

The right upper border is defined by the superior vena cava (1). This is superimposed on the shadow of the ascending aorta (2). It has a light loss of translucency similar to that of the inferior vena cava that is it is slightly less dense than the main cardiac muscle shadow. Below it we find the T shaped shadow of the right branch of the pulmonary artery (3) entering into the indistinct semi-circle of the hilum. Next comes the border of the right auricle (4) leading to the cardio-phrenic angle. This should be easily recognized but not clear-cut with a pronounced edge. In its inner angle we can usually see the downward and outward sweep of the inferior vena cava (5).

At the upper left border we find the aortic knuckle (6), and below it the elbow shadow of the left pulmonary artery (7) then the conus (8) and finally the outer border of the left ventricle (9), leading to the cardio-phrenic angle. In the angle we may see a slight downward and outward loss of translucency comparable to that of the inferior vena cava this is the pleuro-pericardial pad of fat.

In its widest part the normal longitudinal heart is roughly one-third of its width to the right and two-thirds to the left of the mid line of the spine. This is why the blood supply to the right lower lobe is so much more evident than that to the left lower lobe and why this normal difference is so exaggerated in the normal abnormality of the usual scoliosis with its convexity to the right.

The absolutely normal film has a fairly clear triangle of translucency with its apex inwards above the conus. This is the area which is filled in by a line giving apparent continuity between the pulmonary artery and the upper outer edge of the left ventricle when the conus swings forwards in scoliosis.

Normal glands do not appear on films indeed only the tracheo-bronchial group and the broncho-pulmonary group of very small glands are uncovered by normal mediastinal contents. It is unwise to diagnose enlarged glands unless they are undoubtedly very prominent. They can be demonstrated only if they are so placed that they show against lung tissue which they cover and this means that they must appear on the postero-anterior film round the hilum or just under the right upper lobe bronchus between the aortic knuckle and the upper border of the left ventricle.

The Diaphragm

The diaphragm appears as two clear cut dome-shaped shadows one on either side of the cardiac shadow acting as the lower limits to the basal translucency of the lung-fields. The right which rises to the sixth interspace is a little higher than the left usually to the extent of one interspace. The blood vessel shadows may be seen to continue below it, but neither in health nor disease unless they are injected with lipiodol do we see the bronchus in this position.

There are two common findings that must not be read as evidence of abnormality of clinical import. The first is so-called tenting of the diaphragm. A small triangle of short base resting on the diaphragm, and drawn into a rather elongated peak is seen towards the inner end of the mid point of one or other half of the muscle. This is

light pleural reaction in the main fissure and is a common finding with broncho-pneu-

monia even when by the history this condition has been of so low-grade that it has not upset the patient sufficiently to make him seek advice. It remains for a long time or permanently. It must not be read as an adhesion as evidence of lung disease unless definite pulmonary focus is seen when it may be due to contraction of the fibres of the phrenic nerve which has been involved in disease process in some part of its course over the cupola and past the hilum. Such peaking is never then at the position of the main fissure and its commonest cause is apical fibrosis usually tuberculous in origin.



FIG. 35. Showing right lung, blood vessels and bronchi end-on, tenting of the diaphragm and obliteration of the costophrenic angle.

The second is a filling in of the costo-phrenic angle on one or other side by an opaque shadow. This is again a common sequence of a previous pneumonia or broncho-pneumonia the exudate on the visceral layer of the pleura has organized. It is not evidence of present lung disease and of no significance unless a lung focus is seen with it, in which case it is usually evidence of a secondary pleural reaction to a tuberculous deposit.

The Zones of the Lung Fields

The zones of the lung fields are defined in accordance with the diagram in Figure 33.

The upper zones are the areas lying between the upper apices and a line drawn horizontally across the anterior lower ends of the second ribs.

The middle zones lie between this line and one drawn similarly across the anterior lower ends of the fourth ribs.

The lower zones lie between this second horizontal line and the lung bases.

These zones are used to designate the position of any abnormality.



FIG. 36. The azygos lobe.

noted on the film. Each zone should be compared carefully with its fellow on the opposite side before any condition noted is judged as a definite departure from the normal. It has already been noted that disease processes are never exactly repeated in opposite zones and that if this simple precaution is not employed it is possible for the common condition of calcification of the first costal cartilage to be interpreted as a tuberculous focus.

Normal lungs appear as dark areas with many linear markings throughout them due to the shadows of the main pulmonary arterial branches and to supporting structure of the parenchyma. We have seen that the hair-like line of the lesser fissure can appear in the third or fourth interspace; no other part of the pleura or its fissures should appear in an absolutely normal film although it is not unusual to find an azygos lobe in the right upper zone. Here a fine line divides the right cupola by running downwards and inwards to the upper part of the cardiac border at which point there may appear a well marked but small oval opacity which is the azygos vein round which the visceral pleura has dipped during development. This lobe has a light loss of translucency as it contains less residual air than the outer part of the upper lobe.

We have already noted that the radiations due to the pulmonary arterial supply go outwards from each hilum which is itself due in most part of its confused shadow to the main arteries and the pulmonary veins. Along their course and also lessening steadily towards the periphery we may see round opaque shadows of blood vessels caught end-on by the rays as they go backwards. In the same way while we do not see normal bronchi which go laterally we can see the round well-defined circles with clear centres which are the shadows cast by backward going bronchioles (see Fig. 35). They may be of fair size near the hilum and should not be read as cavities or mistaken for primary foci of childhood tubercle which are opaque irregular and hard looking and usually well out towards the periphery.

At the danger of repetition it must be stressed that variations in the normal striations are as much evidence of abnormality as the shadow of the disease process which causes them. If we see in the outer part of the right lower zone the shadow of pleural effusion we must look carefully at the striations in the mid and inner parts of this zone. If the lung has no disease in it we expect to see heavier striations caused by hyperaemia from the

pressure collapse of the parenchyma. If we can see no vessel markings we know we are dealing with a pleural effusion which is secondary to a consolidation of the lower lobe.

The Lateral Film

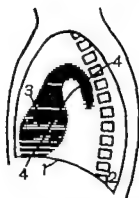


FIG. 37 The left lateral film.

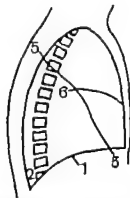


FIG. 37a. The right lateral film.

A lateral film may be taken to give additional information in the localization of lung disease. For example it will throw a shadow that appears to be hilar into its true position in the apex of the lower lobe. In the case of abscess formation in that broncho-pulmonary segment which is supplied by the posterior horizontal branch of the right lower lobe bronchus.

Lateral films may be taken of the right and left lung fields. It must be realized that the shadows of the opposite side although out of focus always contribute to the picture. In the accompanying diagrammatic representation of the left lateral film there can be seen between the vertebral column and the sternum —

- (1) The diaphragm
- (2) The posterior recess of the costo-phrenic angle
- (3) The cardiac shadow resting on the anterior surface of the diaphragm and continuous in its upper limit with the arch of the aorta

The position of the main interlobar fissure is shown as a line (4) running from a point below and posterior to the apex downwards and forwards to a point near the junction of the middle and anterior thirds of the diaphragm. The lower lobe therefore occupies the area below this line.

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On the diagram of the right lateral film the position of the great fissure is shown as the line marked (5) while in addition the lesser fissure is shown as (6), running between the hilum and the posterior border of the sternum. It may appear on the lateral, as it does on the postero-anterior film in the absolutely normal subject.

SECTION II

CHAPTER VI

PHYSICAL EXAMINATION—INSPECTION PALPATION AND PERCUSSION

PHYSICAL examination of the chest has suffered severe criticism in the past few years. This is due mainly to the realization among tuberculosis workers that phthisis can exist for months and even years without producing symptoms and that in its earlier stages it may give no abnormal findings to the most expert examiner while showing definite abnormal shadows on the film. The introduction of mass radiography was urged for these reasons; its arrival has accentuated the criticism. Nevertheless physical examination like bacteriological examination must remain for ultimate diagnosis. The X ray is not the whole patient. In the hands of an expert it can give detail of diagnosis with uncanny accuracy but without serial examinations it can seldom give the answer on activity or retrogression of the disease process. The clinical condition as a whole part of which can be summarized only by expert physical examination can alone lead the physician to an opinion on the present pathogenesis on which he must base his immediate treatment.

Nor must we confine physical examination to any one of its parts. Eyes first, hands next and ears last is still a good guide. It is the writer's experience that many students concentrate too much on the stethoscope and that the statement that there are no physical signs means usually that the examiner has found nothing abnormal to auscultation.

It will be assumed that the student is familiar with all the usual methods; only such necessary aids and such points as the writer has found of help to post-graduates will be detailed.

(1) Clubbing of the Fingers. Inspection of the terminal phalanges should be made in all patients suspected of chest disease, acute or chronic. Clubbing is the phalangeal form of hypertrophic pulmonary osteo-arthropathy which is caused by the forming of a thin subperiosteal layer of bone and thickening of

connective tissue over it. It is found in chronic heart affections and chronic jaundice but is most commonly associated with lung diseases. Toxins and chronic congestion are said to have something to do with it but its real cause is unknown. We do not know why in the terminal phalanges it shows such marked thickening of the soft tissues in some cases which have no apparent difference in their lung pathology as against their colleagues with no finger changes. Its absence then is of no diagnostic significance. Its particular type when present can be so characteristic as to be diagnostic between septic and fibroid diseases between lung abscess and pulmonary tuberculosis.

The two main types show first stage in common. This consists in a filling in of the connective tissue at the root of the nail so that there is no gap between the lunula fold and the proximal end of the nail such as appears on the average normal finger. In addition, this excess tissue is lightly raised and pushing up the lunula fold, is always cyanotic and glistening, as if by continual polishing. While this initial change is later in appearance in phthisis than in the acute septic conditions where as in lobar pneumonia complicating pneumonia it can appear within a few days of the onset it can be very helpful as a hint of the possible presence of symptomless phthisis.

In the further changes associated with proliferative tubercle of adult type the nail becomes humped bowed dorsally smooth and rather pearly-pink, while the palmar surface of the phalanx becomes more and more pendulous and baggy. The finger is thus given a claw like appearance and may even look tapering as the dorsal-palmar alteration is not offset by any marked change in the lateral dimension.

This is entirely unlike the effect of septic disease either acute or chronic as in lung abscess and bronchiectasis where the alteration is generalized over the phalanx. This alteration is in all dimensions gross and dramatic in effect often bulging sharply at the joint with the proximal phalanx, to stand out as an ugly blunt stump. It can come on so rapidly in acute sepsis as to be noted in growth almost from day to day. It can disappear as rapidly after efficient surgical drainage of an interlobar empyema. Naturally this form can slowly replace the more usual tuberculous type in long-standing fibroid cases where bronchiectasis if not true advanced bronchiectasis is a common and expected complication. The patient in this case is suffering more from the

consequences of his disease than from present activity and bronchitis or bronchiectasis is all too easily given as a label to one who is a chronic source of tuberculous infection to all his contacts.

In this connection we may note that emphysema is given as one of the causes of clubbing of the fingers. From our considerations of pathology we shall see that emphysema is almost always a secondary condition. Its primary cause is the reason for the finger changes in the vast majority of cases.

(b) Inspection of the chest as a whole should be made before any palpation methods are employed. Much confusion can result in all examinations up to film inspection if the presence of the normal abnormality of scoliosis is not recognized from the beginning. A general inspection of the chest back and front should warn the examiner of the true condition, and keep him from making some diagnosis of pathological import. It will prepare him also for those changes from the absolute normal which he will see on the radiograph, in the shape and position of the heart, and in the prominence of hilar and large pulmonary vessel shadows.

PALPATION (a) The Sterno-mastoid Sign

Several methods of diagnosing mediastinal shift by assessing the displacement of the trachea have been described. Most of these rely on deep palpation in the supra-sternal fossa, a procedure to which most patients object very strongly. A much easier and more reliable method is that to which reference is made several times in the course of these studies. It consists in gentle palpation across the tendinous origins of the sterno-mastoid muscles at the upper anterior surface of the manubrium.

We have seen in various chest conditions how the apex beat is not nearly so reliable a guide to mediastinal displacement, how the heart swings on its long axis much more readily than it does bodily to one or other side, and how for this reason while it responds to inspiratory movement as shown in screening of cases of atelectasis it does not give at positions of rest the same amount of fixed change to act as a guide to the examiner. Thus it moves backwards (from left lower lobe emphysema) in right lower lobe collapse, and its apex beat to palpation or stethoscope may give no aid to the actual diagnosis while the sterno-mastoid sign will be strongly positive on the right side.

The main value of the sign is seen in cases of unilateral disease: it is only to be expected that bilateral disease will give bilateral



FIG. 34. Positive left sterno-mastoid sign

changes of similar type. The onset of disease in the left side following an established disease in the right, will inevitably undo the value of the sign previously present in the right sterno-mastoid.

In acute conditions such as uncomplicated pneumonia, broncho-pneumonia and lung abscess, as in cystic disease of the bronchi and the epituberculosis of childhood it is negative. We have discussed the reason for this. These diseases do not destroy cartilage and have no effect on the normal pressure as usually exerted by residual air in the alveoli. In chronic conditions with loss of elastic tissue, telecystosis and later with fibrosis it is positive again in tuberculosis and bronchiectasis.

For its discovery the patient should be placed in good light, standing, sitting or lying in a relaxed position with the head central to the thorax. Gentle palpation with the index finger across the muscle tendon is quite sufficient; the sharper edge of the affected tendon which as against its softer, rounder neighbour

will resist pressure is easily felt. With very little practice the observer will see the sign before he applies palpation.

(b) Lung Movement

Many students attempt to assess lung movement by placing the palms of the hands over the mammary regions with the fingers pointing upwards and outwards towards the outer sub-clavicular regions. By so doing they cannot hope to find anything beyond the anterior forward movement of the thorax which is minimal as against the downward excursion of the lungs in response to diaphragmatic contraction and the lateral movement in response to the increased sub-atmospheric pressure of the intra pleural spaces. The assessment of downward movement by percussion will be discussed below. For proper assessment of lateral movement by palpation the palms and closely approximated fingers should be placed gently but firmly in contact with the axillae with as wide an angle as possible between the forefingers and thumbs. The whole hands are then moved inwards still in close contact with the chest walls until the thumb-tips meet in the mid line of the sternum. The patient is then asked to take in a long, slow breath, filling his chest without heaving up his shoulders. Lack of movement on one side as against the other is immediately evident. It will not move the palm and fingers outwards and the thumb on which these act as a lever will move but little from the mid-point as against its fellow. If the method is properly employed expiration should bring the thumb-tips together again at the mid-point of the sternum. The method is much more easy of application posteriorly.

Where bilateral emphysema is present the thumbs move from the mid line hardly at all while the whole hands move upwards rather than outwards with deep inspiration.

PERCUSSION

() General. The note in the lungs is individual and comparative one side with the other. The normal for any patient should be assessed on the finding at the left apex to gentle percussion, with the understanding that while sound enters into such assessment the feeling of resistance transmitted to the pleximeter finger by the underlying tissue is of equal importance. Heavy tapping produces much noise but little to add to the knowledge of the

PERCUSSION

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examiner Grades of departure from the normal can be described as impairment, dullness up to dead dullness, tympany and marked tympany.

A not on the left pex equal to that of the right must lead to a suspicion of left apical abnormality. Its pictorial representation by one line helps us to compare it with more marked abnormalities. These two lines will represent the usual dullness of pneumonia and bronchiectasis, three the underlying resistance of pneumonia and fluid, and four the tonelessness and marked resistance of massive atelectasis and new growth. Tympany is found in the higher pitch and lessened resistance of the basal emphysema of chronic bronchitis and marked tympany in the loss of lung tissue replaced by air in pneumothorax.

(b) Diaphragmatic Movement. Percussion to be complete should include examination of the change of note at the lung bases with inspiration and expiration. A normal diaphragm should move downwards at least one finger. A normal diaphragm should move a half breadth in the mid line of each half posteriorly with deep inspiration. Lessening of extent of such movement with deep adhesion up to complete fixation of the diaphragm will indicate the usefulness of percussion in diagnosis re —

() The dullness of upper lobe phthisis is predominantly posterior. Examples of error we have noted several times how tuberculous in adults is posterior disease. When there is a decision has to be made on physical signs between upper lobe pneumatic phthisis and collapse from mitral bronchial obstruction this finding is a help. Seeing the collapse I have its dullness mainly anterior and most marked about one finger breadth below the true third of the collar-bone at which point we are near the entrance of the episternal bronchus.

(2) A collapsing right lower lobe will show dead dullness in the inner half of the right lower zone posteriorly offset by tympany from emphysema of the upper lobe in the costo-phrenic angle. As against this lower lobe pneumonia shows dullness anteriorly and posteriorly equal at any one level from the axilla to the mediastinum while the dullness of pleural effusion will be axillary and that of collapsing right middle lobe anterior and most marked just below the nipple.

CHAPTER VII

AUSCULTATION

No student can hope to be able to distinguish the various abnormal sounds that can be found by auscultation unless he has accustomed himself to the many variations of the normal as they present themselves to his individual sense of hearing and tone. He should avail himself of every possible opportunity to examine healthy lungs in the fat the thin and the muscular using one uniform rule as his base line—*g* always listening in the upper axilla or to the inner side of the lower inner border of the scapula, while the subject takes long quiet breaths through the mouth.

He will soon find the following three characteristics of the normal —

(1) The sound is a gentle rustle as if made by an infinite number of individual sounds that run together an occasional one of these being slightly louder than its neighbours

(2) Inspiration is slightly longer than expiration

(3) Inspiration fades into expiration with no interval

If now he will listen by placing his stethoscope over the trachea he will find four distinct changes

(1) There is a distinct gap between the end of inspiration and the beginning of expiration

(2) Inspiration and expiration are of equal length

(3) There is an entirely new quality in the sounds. They are pitched higher than normal as if the normal sounds of a large area were concentrated at one point or the patient were blowing gently through a tube

(4) The sounds are louder

The first and second are present in every type of breath sound that is called bronchial breathing the third and fourth vary considerably in different disease conditions all of which are described as giving bronchial breathing

Naturally every teacher demonstrates his own acuity of hearing and sense of tone. The writer has used the following rough rule of thumb —

[Over scattered areas of fibrosis and atelectasis such as are

found in chronic pulmonary tuberculosis and bronchiectasis and over a larger single area of compressed lung (sometimes also a pleural effusion) we get the pitch as described without the loudness. That is *w. h. Pitch +*

II Over a small area, e.g. over the point of occlusion of a bronchus filled with new growth or foreign material and over a larger area of consolidation, as in lobar pneumonia we have comparatively *Pitch ++ loudness +*

III Over a cavity connecting with a patent bronchus we have *Pitch +++ loudness ++*

Here the pitch is so much higher that the listener has the sensation that his patient is now blowing over the end of a tube not through it.

What now follows is built on the writer's thesis that chest diseases produce interference with normal breath sounds by giving extra sounds which can be reduced to a very few basic types and that what denotes the individual disease process producing them is their position of onset and intensity during inspiration. This thesis is built on a conscious effort over twenty-five years of observation to relate internal study of these sounds with the corresponding ultimate diagnosis. It follows that its definitions and consequent statements will to many appear to beg the question, and to others to be dogmatic. They are not meant to be so: they are stated as the writer has employed them in his practice and has attempted to teach them to students.

Every student should acquire the habit of listening intently to the whole phase of inspiration while the patient takes long quiet breaths through the mouth, so that noises of possible nasal obstruction do not interfere. With the first breath he should try to assess the length of inspiration with a view to dividing it consciously if arbitrarily into three parts, 1 which we shall refer

to as the bronchial, bronchial parenchymatous and parenchymatous phases. The writer believes the student will be able to place abnormal sounds in accordance with their causative diseases as

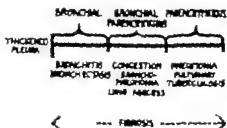


FIG. 39

these affect the bronchial and/or the parenchymatous structures. We hear the sounds known as *sibilli* in both bronchitis and pulmonary tuberculosis but whereas in the former we hear them mainly in the first or bronchial phase in the latter they are mainly in the last or parenchymatous stage. Thus the same actual sound can be of very differing import to the examiner and the patient. We see why it is so easy for a case of active tuberculosis to be interpreted as merely bronchitis.

There appears to be considerable confusion in the minds of many students because they have no clear understanding of the true meaning of the many described abnormal sounds heard interfering with inspiration. They are so subdivided in the usual teaching that they lead to confusion. What is read by one physician as the only true sign of early tuberculosis may be read by another as evidence of a late stage of the disease.

For all practical purposes the writer believes that added sounds can be reduced to three main types each with the particular significance of its underlying cause. They are —

- (i) *Crepitations* fine and coarse
- (ii) *Sibilli* and *rhonchi* (coarser *sibilli*)
- (iii) *Râles* fine medium and coarse (consomating or metallic).

I. *Crepitations*

If the name *crepitation* be confined to a sound which has a dry quality to the ear we can correlate it with fibrosis i.e. increased deposition of connective tissue which we shall see occurs in various diseases considered under the section of applied pathology. The breath sounds are now so changed that their individual components appear to be a series of dry crackles. The coarser these sounds become the less seems to be their number. They will indicate to us permanent structural change which is not removed by cough — a restricting material which lies round the bronchi and attempts to interfere with their natural expansion and elongation in response to the act of inspiration. In the pleural cavity fibrosis is the result of adhesion between pleural surfaces. We can hear *crepitations* therefore at two points in our examination

- (1) First they can appear by the act of coughing. They are

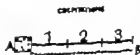


FIG. 40.

- A. With act of cough in pleural thickening.
- B. With fibrosis of lung.

then fine approximating to the sound produced by rubbing the hair about the ears. We are hearing the disturbance of interpleural adhesions which cough produces. They are a common finding in pulmonary tuberculosis and lead us to conclude that there is irritation of the pleura from an underlying lung lesion close to the surface. Naturally once the layers are firmly approximated and no longer at the broad and butter stage of organizing fibrin they will disappear. Their finding can be of significance in prognosis and in treatment. In considering the advisability of inducing artificial pneumothorax.

(2) The comparatively coarser crepitations of peribronchial fibrosis are heard throughout inspiration and do not disappear after cough. They are heard in all chronic diseases from chronic bronchitis to pulmonary tuberculosis as evidence of chronic hyperaemia and the consequent deposition of fibroblasts in the connective tissue. If we listen carefully we may be able to find the part of lung in one particular phase the main trunk of the structure on which the main trunk of the disease is falling. Thus if they are most insistent in the first or bronchial phase falling off in the second and third phase we know we are listening to fibrosis round the larger bronchi in olving surrounding lobules and that the patient has the way open to enlargement of these bronchi up to established bronchiectasis (see Fig 4). This is what experience shows occurs in cases of continuing upper sinus infections which cause constant irritation of basal bronchi.

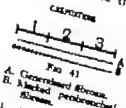


FIG 4

A. Generalized Fibrosis.
B. Affecting Peribronchial Fibrosis.

In the middle or bronchial-parenchymatous phase they indicate that combination of bronchial and lung fibrosis that occurs in lung because of a chronic character. The disease began in the lung. It is now infecting the bronchi that supply the diseased area of lung tissue and we are hearing the resultant fibrosis. In the last or parenchymatous phase they are most intense in the presence of disease that begins in the parenchyma. Such fibrosis is general in lung where pneumonia has healed by epiphragm rather than resolution. In the most localized areas of chronic adult pythias it shows the character that has occurred in the constituents of the affected parts. Fibrosis in these three phases warns us of the danger of developing cavitation in each of them. It precedes the formation of cavitation.

and remains when they appear. As already stated the crepitations are indicative of a change that will not recede, that may be masked by emphysema later but may also lead to much more serious damage if the prime cause continues. If we read into them the lessons they try to convey they will aid us in prognosis as well as in diagnosis: thus their continuance in the mid-phase after the apparent complete evacuation of a lung abscess warns us that our patient is not yet clinically well; that a so-called recurrence of his abscess may follow and that what is really threatening is a consequent bronchiectasis by involvement of the bronchus.

2. Sibili and Rhonchi

These are best interpreted as indicating moisture obstructing the passage of air through bronchi small and large. While very

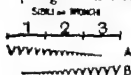


FIG. 42.

A. Bronchitis.

B. Pulmonary tuberculosis.

little moisture in the smaller bronchi will produce sibili; still smaller amounts in bronchi under contraction will give the same sound. The quality of the sound does not differ by the origin of the obstructing material. In other words, we can hear sibili that are due to strip-

ping of the ciliated epithelium in acute bronchitis and sibili that are due to material entering bronchi from breaking down parenchyma in phthisis. Again we hear them in acute asthma, because even the natural moisture normally removed by the action of the cilia cannot be dealt with: the last circular muscular fibres are contracting so severely in the terminal bronchioles.

It therefore we apply our scheme of dividing inspiration into three parts we can correlate sibili and rhonchi with their pathological cause.

In the first phase they will be bronchial in origin: they indicate bronchitis and bronchiectasis.

In the second phase they will be bronchial-parenchymatous. We are listening to diseases which affect both bronchi and lung, e.g. broncho-pneumonia that is resolving, and lung abscess discharging. In the first they will be over a considerable area of the lung; in the second, they will be localized.

In the third phase they will be parenchymatous in origin: material is entering terminal bronchi from the lung, usually from tuberculous deposits that are caseating. Now the sibili and

rhonchi begin towards the end of the second phase and become more and more intense to the end of inspiration

3. Rales

According to the thesis we are following the student should consider that rales mean that there is definite involvement of the lung parenchyma which is breaking down and discharging into a bronchus. They indicate a greater amount of occlusion of the lumen of the bronchus than is present with sibilant and rhonchi. If they are especially coarse and consonating and metallic in character they should be read as evidence of cavitation.

By their position in inspiration we can get a definite aid to the underlying pathogenesis. Thus if they are most marked in the first bronchial stage we can correlate them with bronchial condition, and conclude they are extensions of a disease that has first produced a bronchial and later a parenchymatous pathological change. That is why we hear them in lobules through the penetrated bronchial lumen. They herald the formation of a lung cavity connected with this destruction continues they get more character. Their finding is then diagnostic of bronchiectatic lung cavitation.

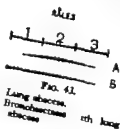


FIG. 42

A. Lung abscess.
B. Bronchiectatic abscess.

In the writer's experience coarse consonating metallic sibilant is a more constant finding than whispering pectoriloquy in all types of cavitation. They can help towards diagnosis when the cavity is comparatively full of disintegrating material and pectoriloquy and loud bronchial breathing may be absent.

Fine rales are heard in the bronchial-parenchymatous middle phase of inspiration in congestive failure. They indicate an inability of the patient to move lobulae.

moisture they are the first signs of that water logging which increases steadily in the chronic bronchitic of gray prognosis. Resolving broncho-pneumonia gives them in this phase in medium intensity they come through to the ear almost as individual small bubbles rising in number.

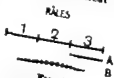


FIG. 44

A. Pneumonia.
B. Bronchopneumonia.

and force to mid inspiration and then falling off again toward the end of inspiration. We are hearing discharging lobules pouring their liquefying pneumonic exudate into the bronchioles.

They are still more insistent and coarser in the discharging lung abscess although they are confined to one area as against those heard in the disseminated disease of broncho-pneumonia. They become more and more consonating and metallic when bronchial blockage has led to breaking down of the lung substance in a case now going on to chronic abscess. We hear now exactly the same sounds we heard in bronchiectasis. There we heard them begin and have their greatest intensity in the first or bronchial phase and they were heard over a fairly large area of the lung. Now we hear them in a localized area and most intense in the mid phase of inspiration. The same pathological process is at work in both conditions. In bronchiectasis the disease progressed from the bronchus into the parenchyma; in lung abscess it progressed from the parenchyma to the bronchus. Chronic lung abscess is only a localized form of bronchiectasis.

In both cases as the disease progresses the râles get more and more metallic in type telling us that communication between cavitation in the lung and an enlarged



bronchus is now established.

In the last, parenchymatous phase we find them in pulmonary tuberculosis. With an advancing lesion they slowly replace the sibil and rhonchi of lesser obstruction while like them they are due to material entering the terminal bronchioles they are now due to actual breaking down of the parenchyma. As the condition goes on to established cavitation the râles get more and more metallic, reaching their crescendo of sound at the very end of the inspiration.

The scheme of interpretation of added sounds is summed up in Fig. 39 which the writer has found useful in teaching on hospital cases for practical demonstration.

Two examples will illustrate diagnosis from physical signs alone —

(1) Chronic fibrocaceous tuberculosis with cavitation of the right lung upper half.

Clubbing of tuberculous type is present in most cases. The right sterno-mastoid is strongly positive. There is marked lack of movement over the whole of the right side. Dullness in the upper

FIG. 45 Metallic râles in tuberculous cavitation.

half especially posteriorly and tympany from emphysema over the lower half

Careful listening to the whole inspiration will present crepitations throughout every phase becoming more and more coarse in the third phase these are from fibrosis At the end of inspiration there will be coarse metallic râles from the cavitation Both crepitations and râles will remain and may be even more insistent after cough With the act of coughing showers of fine crepitations indicate pleural thickening

(2) Acute lobar pneumonia right lower lobe before resolution There is no clubbing of the fingers There is no ternomaxillary sign There is marked lack of resonance over the right side and dullness both anteriorly and posteriorly over the lower half There are no added sounds the breathing is bronchial all over the affected lobe

SECTION III

CHAPTER VIII

SOME GENERAL POINTS AND THE VISUAL, OBJECTIVE DESCRIPTION OF THE ABNORMAL FILM

IN this section we shall gather together such changes from the normal as we shall see in the films of diseases discussed in detail in the section on applied pathology. It is possible to group them under their main characteristics. For example most diseases of the lungs have in common an inflammatory exudate filling lobules as a result of irritation of their alveolar walls. This common process has a common homogeneous X ray shadow. It is denser than that of thickened plastic pleurisy but far less dense than that of atelectasis. It therefore follows that the shadows seen in these three separate conditions are all objectively described as homogeneous, although they may by the experienced eye be correlated with their individual underlying pathology especially if they are studied in conjunction with the clinical details. These should always accompany the request for a report on the film of a patient who has not been clinically examined by the reader. In other words a film report should be in two parts: the first a visual objective description of what is actually seen; the second an opinion on the cause.

It goes without saying that anyone attempting to give an opinion on an abnormal film must have an extensive knowledge of the normal and of all normal abnormalities. Reference has been made several times to difficulties that can arise by the reading of a pathological cause into the normal abnormalities of scoliosis, or simple calcification of the costal cartilages. Even the most experienced observer does not despise the assistance of a good example of the normal.

THE VISUAL DESCRIPTION

Some general rules for guidance may be set down —

(1) Read every film according to a fixed routine such as that given in the chapter on the normal film

(2) In reporting on the trachea, heart and diaphragm not any change in size, shape and position.

(3) In the lung zones look first for the vessel markings and the lesser fissure of the right lung, noting any increase, decrease or change in position such as bunching or splaying of vascular shadows.

(4) Describe abnormal shadows according to their basic types remembering that in front of them, behind them there may be shadows of normal structures. They may be considered under five basic types: streakiness, homogeneous shadows, non-homogeneous shadows, ring shadows and military shadows.

Streakiness is the name applied to increases in the normal lung markings.

Homogeneous shadows are those which do not alter in their evenness throughout their extent. Although they may differ in density they do not do so suddenly and interruptedly.

Non-homogeneous shadows are those that alter suddenly in density at one or more points throughout their extent.

Ring shadows have more or less circumscribing limits. In their centres they may have homogeneous or non-homogeneous shadows, areas of translucency. Similarly their limiting boundaries may consist of one or other of these types of shadows. These shadows will be described in more detail in the next chapter.

(5) Give the size and shape of individual shadows: they may be single, multiple, large or small, well-defined, diffuse, along the lines of normal markings, confluent or patchy, round, oval or triangular.

The term "mottling" is often used in descriptions of shadows. It conveys the idea of mixture of opaque and non-opaque areas. In this sense it can apply to such shadows as occur in the film of uncomplicated broncho-pneumonia. It is however often used to describe a mixture of homogeneous and non-homogeneous areas such as are found in the film of pulmonary tuberculosis and some interpretations confine it to this meaning. It is therefore not a good word to use as being in the literature half descriptive and half interpretative and with no accepted definition under either heading.

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CHAPTER IX

THE INTERPRETATION OF ABNORMAL SHADOWS. STREAKINESS HOMOCENEIOUS AND NON- HOMOGENEOUS SHADOWS

BEFORE giving an opinion on the diagnosis of a patient under review the examiner should now inspect the film more closely at about 1 foot from the viewing box and then go back slowly to a distance of some 5 or 6 feet. The writer realizes that this will not make the film stereoscopic what it will do is to make individual more opaque areas within a shadow that looks more or less homogeneous on close inspection stand out against any background common to them. Unfortunately this point cannot be demonstrated by prints but only by the study of films on viewing boxes.

There will always be the difficult cases where shadows are so confused or ill-defined that their underlying pathology cannot be diagnosed. There is no such thing as the typical film for every disease there are always individual differences. Furthermore, we all understand that X ray examination is at best only an accessory to general and specialized physical and laboratory investigations. Consideration of the general condition, the temperature the blood-count cellular content of effusion and the result of bronchoscopy and biopsy may have to come into the summation of the interpretation together with serial film examination, before we know in any one hypothetical case whether we are looking at a lobar atelectasis which is basically post pneumonic post-operative or due to carcinoma of the bronchus. There are, however from the experience of various disease conditions as reviewed under applied pathology certain guidances to be had if we look now more closely into the main types of shadow defined in the last chapter. It is understood of course that while these are read individually their accompanying shadows and the evidence of interference or non-interference with other structures of the thorax are read with them for interpretation. Thus the shadows of pneumonia and of atelectasis of the right upper lobe are

both homogeneous in type but we have seen that there is no change in the blood supply of the middle and lower lobes in pneumonia, whereas in the latter there is a playing of the blood supply since they have become emphysematous.

Streakiness and Absence of Normal Markings

Decrease and increase of normal lung markings are equally important. Decrease is found in pneumothorax, cystic disease of the bronchi and emphysema. In pneumothorax the loss of markings appears outside the line of the collapsed lung, while this is offset by increased markings within it. With cystic disease some faint markings of normal lung in front of or behind the cyst may occasionally be seen but usually there is an inscribed area of the translucency of pneumothorax. Often there are with larger cysts definite increased markings outside the cyst due to pressure collapse of surrounding lobules and the crowding together of their normal structural connective tissue and blood supply.

With emphysema there is decrease of small or large areas of the lung, giving an increased translucency of the affected part. Where considerable portion is affected as in the gross bronch emphysema of chronic bronchitis or in the costo-phrenic angle emphysema of the upper lobe and the lower lobe telecystosis only the faint markings of the supporting structure of much-distended lobules can be seen. Such a film diagnosis of emphysema may be of great import to the patient. As I have stated if it is not accompanied by apparent X-ray cause the ordinary film examination. It may call for special examination by lipiodol injection or bronchoscopy.

Increased streakiness is found in hyperaemia. We shall see how hyperaemia is the first indication of acute bronchitis and the constant accompaniment if not the basic cause of chronic bronchitis. We have seen too that we ought to look for hyperaemic markings in a lung under collapse from pleurisy with effusion and how we must suspect consolidation of the lung if we do not see them.

The distinctive feature of all shadow connoting fibrosis is that they stand out in increasing relief if we observe the film as we go backwards to some 5 feet from it. This characteristic they share with those shadows we have named non-homogeneous. In chronic bronchitis they are almost invariably along definite lines as indicating that there is increase in peribronchial connective tissue caused

by the hyperæmia of its vessels. With fibrosis we may see corroborative evidence of the true pathology in disturbance of the normal position of other recognizable structures e.g. change in the position or density or both in the lesser fissure rib of the diaphragm and perhaps displacement of the heart and mediastinum in such a disease as bronchiectasis. Again we see streakiness perhaps dissociated from the usual normal markings in carcinoma, especially in the nodular medial and basal types. We find it also with or without discrete nodules in the reticulations of silicosis when we are viewing the congested and thickened lymphatics associated with this disease.

Homogeneous Shadows

These shadows when in the lung result from exudates and de-aeration. In the pleural cavity they are due to exudates and occasionally to transudates.

On the whole it can be taken as a safe rule that homogeneous shadows indicate acute lung disease. They are found in pneumonia, broncho-pneumonia and in the acute progressive parts of tuberculous lesions. Their variation in density can give us a hint of their underlying pathology. Homogeneous loss of translucency of lesser density is found as the X-ray evidence of inflammatory reaction in the alveoli. This is due to irritation of the walls of the cells by the organisms which later penetrate the walls and fill the cells with exudate. Such exudate throws a denser but still homogeneous shadow.

It follows that under this type of shadow falls that called by the name of pneumonia. This term is justifiable if it is merely descriptive and not meant to be specific and interpretative. It is not a pathological entity. It is the shadow common to every process irritative to lung parenchyma, being the oedema or catarrhal exudate seen in pneumonia, broncho-pneumonia, lung abscess, bronchiectasis and tuberculosis. Its presence means that some specific disease is at work, e.g. It may be the shadow of fleeting type such as we see in tropical eosinophilia. In every case we require serial films and we may require lateral and tomograph examinations and even lipiodol injection or bronchoscopy to reveal the underlying cause.

In uncomplicated pneumonia and broncho-pneumonia we see a homogeneous shadow involve the broncho-pulmonary areas of a main bronchus or one or more of its branches. In phthisis we

HOMOGENEOUS SHADOWS

see it in the localized rows of dorsal bronchioles. In both we can follow its progress from the catarrhal to the specific heavier shadow so that we are now able on the film to recognize the advancing edges of the disease process. Similarly we may note the first loss of translucency in retraction which comes with the absorption of oxygen and trace it to the last which is due to the actual invasion of the parenchyma by carcinomatous growth. Thus the last and densest stage is most evident sometimes from the beginning as the site of bronchial blockage.

On the pleura we recognize the first generalized fine loss of translucency that comes with pleural pleurisy so often the back ground to a tuberculous lung lesion. To an underlying lung abscess. The same fine loss of translucency is found in the shadow of the opaculent fusion of fibrin throughout it and in especial density with the organization of fibrin against the lung which is under on the visceral layer of the pleura against the lung which is under pressure collapse. If the fusion becomes localized and begins to organize in the pleural cavity it gets more and more opaque until it reaches the opacity of pneumonic exudate in the lung. We have then the density of the shadow of pneumonia.

All homogeneous shadows share one particular they do not all arise in their similarity when viewed at a distance. The shadows of pneumonic exudate in pneumonia broncho-pneumonia and lung abscess, are homogeneous in this sense. The wall of a proliferating tuberculous cavity will see a never homogeneous. With two examples we shall be able to illustrate this point more fully.

In broncho-pneumonia we can follow the progress of the disease in shadows first evident near the upper halves of the right auricular or left ventricular border of the heart, going then outwards and downwards along the lines of the main blood supply to the lower lobes. In the developed lesion we recognize along these lines, homogeneous triangular like areas of equal loss of translucency near quite discrete but never quite confluent because between them are areas of inner homogeneous loss of translucency. The first shadow is close to pneumonic exudate the second to the non-specific inflammatory catarrhal exudate. Neither changes in type or sharpness by distance as against near viewing. This is because we are looking at material on the lung lobules that have not organized. There is no collapse of lung cells. We have corroborative evidence in the absence of mediastinal shift

CHAPTER X

ROUND SHADOWS

Ring shadows may circumscribe areas of comparative density or comparative translucency. The ring formation may be of homogeneous or non-homogeneous type whether it is thin or heavy in outline.

() Ring Shadows round Areas of Comparative Translucency. The thinnest of all homogeneous boundaries is found in emphysematous bullae as here it consists only of the visible strands of stretched supporting tissue round an area in which no connective tissue streakiness is seen either because normal tissue is stretched or broken beyond recognition or because the tissue is absent by congenital abnormality. We see such areas particularly towards the upper and upper-out portions of the upper lobes and along the inferior and lower anterior margins of the lower lobes which areas are naturally weak in connective tissue. We recognize these bullae sometimes in the lung of a child who has had severe whooping cough often in the chronic bronchitic towards the bases and occasionally in the congenital and so-called essential emphysema of unknown recognizable origin.

These bullae do not look in anywise different from the type of ring shadow sometimes seen in known cases of pulmonary tuberculosis the writer has watched many of them come and go in that type of case which seems to have more than ordinary pleural reaction to the underlying disease. Some disappear altogether as quickly as they appeared but many leave in their low zones a ragged fibrotic looking shadow which now produces in previously silent areas those added sounds which we associated with pleural thickening; bursts of fine crepitations with the act of coughing. They would therefore appear to be the localized emphysematous bullae localized pneumothoraces over small lesions of the lung surface.

A more visible but still homogeneous outline rounded rather than oval is seen in congenital cystic disease of the bronchi. These cysts may be single or multiple separate or apparently

which shows that these materials are replacing the residual air and not altering the normal pressure which residual air maintains.

Again in the film of adult proliferative phthisis we have, at some stages of the disease when exacerbations are taking place, the possibility of three homogeneous shadows. First there is a generalized fine loss of translucency forming a background to all other shadows: this is the plastic pleurisy of slow irritation and adhesion of the overlying layers of the pleura. Second we see scattered areas on the edges of the main shadows: they have a density slightly greater than the first, and equal to that of the catarrhal exudate of broncho-pneumonia. Next we see the denser homogeneous shadow of tuberculous exudate. In lobules where the tubercle bacilli have invaded bunches of acini to produce true tuberculous exudate. None of these alter materially on distant viewing while all three incline to merge when we look very closely. The distant view will however throw into strong relief any non-homogeneous shadows also present.

Non-homogeneous Shadows

These are shadows which alter materially in their density within their extent on viewing at ordinary distances and still more so when viewed at a greater distance. As against homogeneous shadows which mean acute lung disease we can take it as a safe rule that they mean chronic disease—bronchiectasis and pulmonary tuberculosis being the commonest. In other words, we can correlate them with fibrosis and organization, and with the type of atelectasis which results from these two processes. They are denser, more clear cut and harder looking and usually very irregular in outline. They may on close inspection appear to be confluent with the rest of the abnormal shadow of which they are part but on distant viewing they stand out sharply. This is because they are surrounded by lobular complementary emphysema, the continual accompaniment of atelectasis in all its forms. We have already noted that there can be no collapse in either small or large areas of the lung, without corresponding emphysema. The translucency of these emphysematous lobules acts as a foil. It offsets the organized areas. It is therefore possible to write an interpretative report of tuberculous infiltration, fibrosis and pleural thickening.

more common in the lung abscess which is not evacuating freely because the bronchus connected with it is blocked

(b) Round Shadows of Areas of Comparative Density

An abnormal shadow may be rounded because of loss of translucency of comparative density. The Aschmann's focus is a lung abscess in the lower part of the bronchus. It is a homogeneous rather fluffy shadow but on distant viewing stands out as a whole against its surrounding tissue. On serial



F 46 Aschmann's focus in right upper lobe

films it may remain unchanged to outside but perched. When it breaks down it occasionally does not quite like the shadow an exceedingly clear but usually homogeneous shadow. It has the semblance to a round shadow being dragged out into the bronchi long exaggerated blood vessel markings of the inflamed bronchi long which the contents of the non-infected parenchyma are attempting to drain to the glands. A distinguishing feature is the

superimposed (see Fig. 9). They may have walls varying in thickness but seldom of more breadth than a 1/16-inch on a 15-by-12 inch film. They may contain a fluid level. If connected with their originating bronchus they can share in bronchial affections up to the sepsis of bronchiectasis and can sometimes be recognized in a collapsed lobe. Ordinarily however in their uncomplicated state they have two definite characteristics in addition to those stated which can aid us to give an interpretative reading. First they have no associated evidence of fibrosis round them second they have no associated mediastinal shift.

The outline of lung abscess which is discharging gives the next more evident homogeneous ring shadow enclosing an area of comparative translucency. The boundary is now round 1/2 to 1-inch in depth. It usually has a generalized hazy loss of translucency external to it and over its enclosed lung tissue this being due to thickened pleura and on its outer edge to catarrhal exudate in those alveoli which have been irritated but not actually involved in the disease process. The actual boundary is made of pneumonic exudate and so does not alter materially in whole or in part with distant viewing as against closer inspection. It has no surrounding fibrosis. It is therefore in every way a single entity in the lung field oval rather than rounded. Its homogeneous character tends to remain up to its change to the secondary stage of bronchiectasis, its inner pneumonic constituent appearing to be frozen in its even while its outer lining shows increasing and decreasing densities with advancing invasion of its sepsis into the surrounding lung. Moreover it still remains an entity and not just part of a generalized involvement of the particular lobe or of the lung field in which it is situated as is nearly always the case with the tuberculous cavity. Apart from the acute excavation of an Aschmann's focus a tuberculous cavity is seldom without definite surrounding fibrosis.

The average tuberculous cavity can be visualized from what has been already described (see Fig. 103). Its surround in both internal and external borders is irregular and uneven. It merges in its outer zone with evidence of fibrosis. This fibrosis with organized and collapsed lobules that are fused together and opaque form its wall which stands out in bold relief to distant viewing. It is seldom the only evidence of disease in the lung. It may contain a fluid level which shows as a homogeneous shadow with a straight horizontal upper border but a fluid level is much

will therefore be the associated fibrosis in and around its supplying bronchus.

The Aschmann's focus is therefore never like the cyst in congenital cystic disease which is affected by acute bronchitis the cyst does not stand out so clearly against its background and as the infection subsides its wall returns to the characteristic thin outline with no resulting fibrosis round it.

There is a type of nodular carcinoma which starts in a small bronchus and involves the parenchyma through the bronchial wall to produce a single rounded shadow. It can resemble an Aschmann's



FIG. 47 Nodular Carcinoma.

focus so closely that it may be impossible to diagnose it with the fullest radiological examination. Only serial films may eventually give the true diagnosis as happened in the case illustrated. Usually however such a shadow is basal and though it is mainly homogeneous is much more dense than the Aschmann's focus and has definite streaks connecting its inner border with the hilum.

An acute lung abscess, often oval rather than rounded has not the clear outline of the Aschmann's focus and does not stand out against its background on distant viewing. Seen just after its

The hydatid will be found to collapse with lung tissue while the others retain their previous relative position to the mediastinum and the bony thorax.

A large calcified tuberculous focus is unlikely to cause difficulty as calcified hilar glands will be evident.

Serial films will differentiate against an Ascarum focus to a lung abscess. During the period when we expect changes in the lazily outlined abscess and in the leaf-cut Ascarum focus which began by standing out against its background on distant viewing the hydatid will cast the same dense homogeneous and rounded shadow.

This same characteristic of remaining static in size and density for considerable period will aid in diagnosis of multiple hydatids against the shadows of metastatic growths if these are confined to the right lower lobe. They increase density with age differ in size and grow steadily. Usually they are in large numbers which is diagnostic feature. The miliary appearance they can produce when they suddenly appear in both lungs is considered in the next chapter.

The conjunction of tuberculosis and diabetes can give many round shadows. Two points aid in diagnosis they stand out well on distant viewing against surrounding emphysema and they are confined to the upper and mid zones.

right lung and in its lower lobe. It can vary considerably in size. Naturally if hydatid cyst is suspected precipitin complement fixation Casoni and blood tests must be done the majority of cases will give a positive Casoni and an eosinophilia.

Multiple secondary hydatids occur usually in the right lower lobe and as they are common results of rupture of a sub-phrenic abscess the liver must be examined and special attention paid to the shadow of the right diaphragm. This shadow will often be raised or dome-shaped or indefinite in outline.

As against the single hydatid we may have to consider the rare true benign chondroma and the occasional neuro-fibroma and dermoid which appear to be in lung tissue. This is because all



FIG. 49. Dermoid of Lung.

three may be rounded, clearly defined, dense and homogeneous. While the chondroma goes on to hard central areas which are very dense by ossification of the cartilage remnant from which it developed as against the peripheral density of the hydatid it may be necessary for differential diagnosis to induce pneumothorax.

The hydatid will be found to collapse with lung tissue while the others retain their previous relative position to the mediastinum and the bony thorax.

A large calcified tuberculous focus is unlikely to cause difficulty in calibrating hilar glands will be evident.

Serial films will differentiate against an Ascarum focus or lung abscess. During the period when we expect changes in the hazily outlined abscess and in the clear cut Ascarum focus which began by standing out against its background on distant viewing the hydatid will cast the same dense homogeneous and rounded shadow.

This same characteristic of remaining static in size and density for a considerable period will aid in diagnosis of multiple hydatids against the shadows of metastatic growths. They are confined to the right lower lobe. They increase in density with growth in size and grow steadily. Usually they are in large numbers which is a diagnostic feature. The milky appearance they can produce when they suddenly appear in both lungs is confined in the next chapter.

The conjunction of tuberculosis and diabetes can give many round shadows. Two points aid in diagnosis they stand out well on distant viewing against surrounding emphysema and they are confined to the upper and mid zones.

CHAPTER XI

MILIARY SHADOWS

Numerous or uncountable areas of loss of translucency can appear in certain or in all zones of the lung fields. Interpretation of the underlying cause depends to a very great extent on the quality of the film especially on its penetration. This is one reason why there is still such variance on the diagnosis of reticulation, which may be read as present in a soft film and be entirely absent on a hard film. Any film of any diseased condition is necessarily like the film of a normal lung a photograph of superimposed structures so that we may be quite unable to differentiate miliary shadows unless we know the patient's past history and in particular his occupational history. Indeed without serial examinations we may even then fail to make a satisfactory differential diagnosis as between miliary tuberculosis, silicosis and siderosis. With this caution, however we may find it possible to give a reasoned opinion if we take the following five criteria in conjunction: the predominant distribution, the size and shape of individual shadows, the density of these shadows, their type of background and the clarity with which they stand out against that background.

Only some of the more usual conditions will be considered. Such conditions as byssinosis, siderosis and asbestosis are too specialized to warrant discussion here.

Acute Miliary Tuberculosis

The acute form of miliary tuberculosis gives a standard against which other conditions may be considered. Both lung fields are covered in every zone from apex to base with dots which vary in size and shape because they are photographs in different planes of the catarrhal reaction to microscopic deposits of tubercle. They are usually at most three millimetres in their largest axis. Individual shadows have a light loss of translucency but are often so crowded together that no lung markings are visible with the result that their background appears as a dirty off white discolouration.

MILLIARY SHADOWS

tion. This background darkness tends to obscure films when the individual deposit coalesce in some places and appear as more ill-defined smudges with superimposed lines.

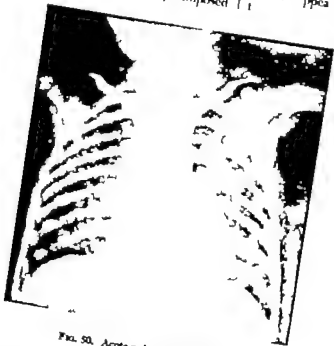


FIG. 50. Acute miliary tuberculosis

Chronic Miliary Tuberculosis

The chronic form of its early stages may defeat differential diagnosis as against the acute form on single film examination alone. Its usual age incidence is of adolescence and early adult life and the latency of the disease must be taken into consideration. Of patients who survive some show progressive clearing of all the lesions others show gradual lessening in the number of deposits those remaining being scattered in all zones but tending to show preference for the upper and mid zones. They are dense and sharp in outline. Usually the background is more translucent than normal but there may be increased striations of fibrous round both hilar regions and over the upper poles. In several

cases there is marked enlargement of hilar glands usually more evident on one side. This stage of the disease is better described as calcified miliary tuberculosis.



F 31 Healed miliary tuberculosis.

Bronchiolitis.

This condition is a blockage of the fine bronchioles as a result of their inflammation. It can occur in children with measles or at any age as complication of influenza or of poisoning by irritant gases such as phosgene and chlorine. When the blockage is complete there is a resulting collapse of bunches of air cells which they supply. If the air cells re-expand as a consequence of the clearing of the bronchioles no ultimate damage to the parenchyma will result. It may be that in some cases in children the recovery is more apparent than complete and that this produces a cylindrical enlargement of bronchi which is later interpreted as congenital in origin. Where measles is the prime cause the majority of cases show a marked coincident enlargement of hilar and bronchial glands.

The predominant distribution of the shadows is in the lower zones falling off distinctly into the mid and upper zones even in extreme cases. There is a similarity to the film of cut miliary

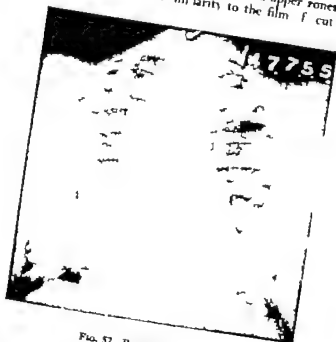


FIG. 52 Bronchiolitis

tuberculosis in the loss of translucency and indefinite outline of individual shadow but careful inspection shows that they tend to be larger and to coalesce because bunches of alveoli are involved in the process. The occluded bronchiole can usually be distinguished as a very fine but definite dot in the centre of the bunch. By film alone the condition may be impossible to differentiate from the results of inhalation of paraffin particles from long-continued nasal medication of oil. All three conditions usually wreck survivors who have inhaled oil.

This is not the case with bronchiolitis obliterans where we see a permanent condition consequent on the inhalation of irritant gases. Inflammation has proceeded to fibrosis within the bronchioles which do not recover. Blast injuries during the war produced

an exactly similar picture. Bronchiolactasis is a very common end result. The distribution is similar to that already described.



FIG. 53 Same patient as Fig. 52 six months later

but individual shadows are much more clear cut. A permanent organization of the nodules gives a more opaque shadow in which we can find no central dot.

Broncho-pneumonia

Broncho-pneumonia is unlikely to cause difficulty owing to its predilection for the lower zones to its tendency to follow the

lines of the bronchi and to its comparatively rapid changes on serial films between the hilum and the periphery

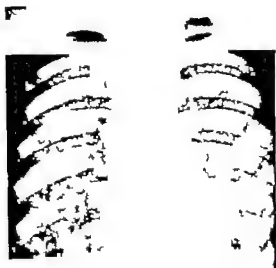


FIG. 34. Bilateral lower lobe broncho-pneumonia.

Silicosis

Medical literature has contained many discussions of reticulation which is stated to be the first stage of exposure to small amounts of silica causes little tearing of the lymphatics which are therefore eliminated. The result is blockage of, and this appears first at the bifurcation. The film now shows a fine fibrosis

late on
L

MILIARY SHADOWS

dots of silica accumulated at the bifurcations. Experts say that it is the composite photograph of these dots taken in superimposed planes of the lung which can give on a soft film that first abnormality known as reticulation.

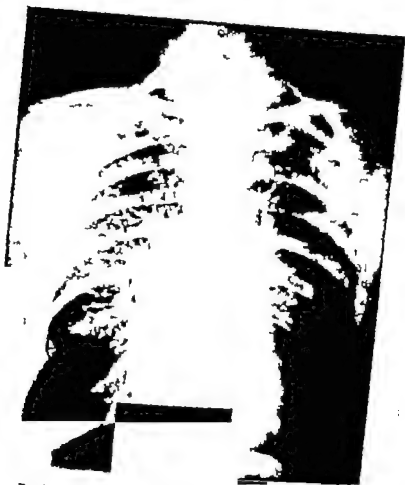


FIG. 55 Reticulation and early coalescence in haematite miners

When definite miliary shadows appear dense and well defined nodules are seen standing out clearly against a background of

emphysema. Such a stage is occasionally reached quite rapidly so that there may be extreme difficulty in differential diagnosis as against one stage of chronic or of a calcified miliary tuberculosis.



FIG. 54. The nodular stage in hematitic miliary

Serial films will then give the answer because the nodules will enlarge rapidly to a size much beyond that of tuberculous deposits and will coalesce into irregular densities. Round these will appear much fibrosis and marked emphysema and individual nodules may be no longer recognizable.

In the usual course of the disease distribution of nodules is a good guide to diagnosis. They first appear in the periphery of the upper lobes. As they gradually increase in number they appear more and more towards the upper mediastinum. In the posterior surfaces of the upper lobes and in the posterior apices of the lower lobes. As we have already noted they have a predilection for the same dorsal branches of the bronchi as the deposits of phthisis.

Anthracosis

Anthracosis or coal miner's black lung is a condition closely allied to silicosis. Indeed it may be extremely difficult to differentiate between them. By long exposure to small amounts of silica



FIG. 57. Anthracosis, early stages.

In the coal dust the blockage we have noted in silicosis takes place at the bifurcation of the smaller bronchioles. Beyond this point carbon accumulates until it fills and later distends the surrounding air cells. When present this localized distension which can be

up to some five millimetres in diameter is a characteristic film. Density of individual shadows varies considerably, some nearest the film appear quite hard. Apical and basal emphysema is said to be a characteristic finding.



FIG. 38. Anthracoosis with typical emphysema.

Mitral Stenosis

The appearance of miliary shadows in mitral stenosis is of grave significance as they are evidence of a failing heart. Individual shadows are rounded and homogeneous (the loss of translucency). Close inspection of the film will show that they lessen steadily in every direction from the hilum to the periphery because they are the shadow of blood clots seen end-on. The hilar shadows are confused and swollen and contain an occasional dense shadow thrown by the principal blood vessels. The marked loss of translucency in the mediastinum gradually merges into generalized lighter loss of translucency due to heart-failure in which are filling the alveoli.

The condition may cause difficulty as against one stage of a miliary tuberculosis or occasionally of an early pneumoconiosis.



FIG. 59 Advanced mitral stenosis with miliary shadowing.

but the dense hilar shadow and the gradation in size of individual shadows which are less clear-cut in outline because of their dark background will generally help in differential diagnosis.

Malignant Metastases.

Blood borne metastases of miliary form from a primary growth outside the lung are rarely seen. The individual shadows are usually ill-defined blotches of irregular size and density. Occasionally they can resemble miliary tubercles or the nodula stage of pneumoconiosis but they are usually more ragged in outline, tend to increase more rapidly in size and to concentrate from above down and so that the apices are comparatively clear. Their sudden appearance and their failure to stand out on distant viewing because they have no background of emphysema, will also aid in differential diagnosis.

While this form is practically unknown as a result of primary lung malignancy, a growth may remain unsuspected in one of the

larger bronchi until it has reached the thoracic duct through the glands and flooded all the pulmonary lymphatics with malignant cells. The lung then appears to be covered from apex to base by a loosely woven fine hair net. This is due to thickened lymphatics where they cross or appear next the film end-on. We can see tiny nodules. If a large mediastinal gland is seen this will help in diagnosis. The condition is known as lymphangitis carcinomatosa.

CHRONIC BRONCHITIS

and get more and more marked until its end

If the condition is very acute and so produces a heavy viscid exudation this may be seen on the film in larger bronchi as loss of translucency running between thin ill-defined parallel lines. The loss of translucency is due to the exudate on the mucosa and in the lumen of the bronchi. It is even and homogeneous in type. The lines are the congested vessels of the bronchial blood supply which lie in the connective tissue round the walls. They are seen running downwards and upwards towards both bases in the lower zones as bronchitis is mainly a basal disease. We cannot tell they are bronchial shadows as we shall remember from our applied anatomy that as against the blood vessels they keep the same width for some distance and within that distance have no branches. Usually they are not well defined except near the hilum and they do not continue below the diaphragm as vascular shadows do even with long-continued irritation that is producing the complication of fibrosis. As the patient recovers from mild acute bronchitis these shadows disappear and the film turns to normal. There has been no permanent damage to the bronchi the hyperemia of the bronchial blood supply has not lasted long enough to bring the fixed changes we find in chronic bronchitis.

From their origin it follows that congenital cysts of the bronchi can share in the inflammatory exudation of acute bronchitis.

Sometimes we see distinct increase in the hilar shadow. This is due to hyperaemia. We shall remember that the hilar shadow is due in the main due to blood vessels. This increase disappears as the patient recovers. If he has the common type of scoliosis with convexity to the right this reaction usually appears more intense and is evident for a longer time than in the absolutely normal subject in the left hilar region.

Chronic Bronchitis

This condition is especially associated with any prime use of increased blood supply to the lungs. This is why it is so commonly associated with chronic hilar disease of the heart. The continual congestion has two effects on the bronchus —

(1) It contributes to the considerable hypertrophy of the mucous membrane. The cylindrical epithelium is destroyed and replaced by cubical epithelium so that there is much less viscid sputum than in the acute form but on the other hand in this process the

cilia which should sweep out the debris in the lumen, have been lost so that there is much more obstruction by material which is now mainly pus from degenerated polymorphs. We see therefore why it is that the abnormal sounds of rhonchi are much coarser than those we heard in acute bronchitis but also why we get from less viscid sputum less loss of translucency within the lines of more inflamed bronchial walls.

(b) It causes small round cells to invade all the bronchial coats from the mucosa outwards. These cells become infiltrated by capillaries and the consequent deposition of fibroblasts with endarteritis of the capillaries leads to increasing fibrosis. The main brunt falls on the connective tissue coat because of the long continued hyperæmia of its blood supply.

On the film we can recognize it in definite white streaks along the lines of the affected bronchi continuing more clearly and much further outwards from the hilum than any normal striations we may see connected with vascular markings or with the supporting structure of the lung. They are particularly evident along the inner bronchial bundles of the lower lobes on both sides only the fact that the heart shadow covers less of the right lower zone makes them more apparent in this area as against the left lower zone. If we stand some distance from the film we can see these streaks give much more evident outlines to the affected bronchi.

Fibrosis gives us a new stethoscopic sign beyond the rhonchi of bronchial obstruction. We hear dry crepitations throughout inspiration which remain after cough. These show us that

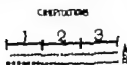


FIG. 61

- A. Generalized fibrosis.
B. Marked peribronchial fibrosis.

fibrotic material round the bronchus is resisting its attempt to elongate and open up by the act of inspiration. What was previously only a supporting surround expanding and contracting with the normal movements of the bronchi, is now constricting band. As this material is static and not movable as

mucus by cough it is a steady finding in all chronic inflammatory diseases of the chest. In its turn it produces emphysema which we shall discuss a little later.

A still further effect is shown on the film (of the advanced case) in an alteration of the normal cardiac outline due to resulting hypertension. An early sign of this complication appears to be an

Increasing size of the aortic knuckle similar to that found in the arteriosclerosis of advancing years. Later comes unfolding of the aorta, where this increase of the knuckle is continued downwards and inwards as a band of loss of translucency which forms the bulge of the *corvus*. Still later comes flattening of the diaphragm which becomes more and more ineffective in its movements from advancing emphysema. Experience shows this warrants a grave prognosis indicating that we may shortly see a protruding superior vena cava and the outward displacement of the right auricular border that herald congestive failure.

Emphysema

The name emphysema is applied to what is essentially a distention of the alveoli. Many types are described in medical literature. For all practical purposes in their immediate and ultimate effects



FIG. 62. Segmental collapse of the right lower lobe as broncho-pneumonia. The line of the collapsed fissure can be seen in the upper part of image. Just inside the distal edge of the collapsed segment there is a small wedge of the costo-phrenic angle.

on lung structures and through this on the patient two types may be distinguished temporary and atrophic

Temporary emphysema occurs with temporary collapse in other words the distension of the emphysematous alveolar walls is complementary and for a considerable time there is apparently as judged by after results no evidence of damage by the stretching of the walls and of the interlobular connective tissue which return to normal. Such a condition is found with temporary blockage of the bronchi small or large. We shall see it can occur in localized form in the ephemeral collapse that often complicates broncho-pneumonia (see Fig. 62) and in the lobar collapse by foreign body in a main bronchus as in the condition of so-called post-operative pneumonia.

The atrophic form is associated with destruction of alveoli breaking down of their walls irreparable stretching up to tearing of their elastic tissue and disintegration of the binding septa which knits them into lobules and links them for support with the peribronchial connective tissue. It results from two prime factors first a weakening of the wall that occurs in all chronic illnesses such as bronchitis bronchiectasis and tuberculosis and second a distension from continued cough that destroys alveolar elasticity by the constant stretch of forced inspiration and expiration.

There is an intermediate form that occurs in generalized fibrosis, which produces a generalized but slight complementary distension of lobules from shrinkage of their supporting connective tissue. We shall note later how this occurs in pneumonia which heals slowly and badly by repair instead of resolution known as post-pneumonic fibrosis the condition commonly affects the whole of one lung. The contracting peribronchial tissue drags on the septa of the lobules bringing a generalized stretching of the elastic tissue of the individual alveoli but it can also become so much thickened that it crushes and destroys the alveoli in its neighbourhood. In other words we have both complementary and atrophic types.

All forms of emphysema occur most easily where the supporting tissue is normally weakest. This is why the lung bases so easily blow out in chronic bronchitis why the lower anterior edge of the right lung so readily distends when the middle lobe collapses to fill in the space vacated by the receding lobe. It is the reason too why with the collapse of one lower lobe the other one so

readily bulges across the antero-mediastinum in an attempt to take its place. The lack of support is the cause why we occasionally see localized emphysema over the plicae of the lung that may rupture without obvious cause of strain and present us with simple spontaneous pneumothorax through leakage of air into the pleural cavity (see Fig. 12). It is this same natural weakness that will cause the outer and upper apical part suffer first in blockage of the upper lobe bronchus; their lobules collapse very easily and so lose their residual air. Thus it is sometimes possible to see slight loss of translucency of even, homogeneous type in the outer infra-clavicular region quite early in case of carcinoma of the upper lobe bronchus.

We have already learnt how elastic tissue allows expansion of the lung, as it responds to muscular stretching throughout the bronchial tree, and how it finally confines the extent of such expansion by its network of terminations round the alveoli. Now the action takes place when emphysema is present. Affected parts have lost their power of movement as they are constantly at full distension. They can be moved only bodily, being dragged by other unaffected lung structures. Hence the heaving non-latral movement of the lung bases in chronic bronchitis and the permanent hyperresonance from constant expansion, which has a tympany definitely beyond that of forced inspiration in normal tissue. Prolonged expiration if it is to be expected from the sufferer nature is attempting to force air out of the blown-out vesicles. We see why expiration is now equal in length to or longer than inspiration.

Blood vessels in the alveoli and in the supporting connective tissue of lobules are drawn out into thin wavy structures and some are occluded together with two results. First there is occlusion of the capillaries; the septa cannot live without blood supply and so break still further. Second the narrowed vessels in the alveolar walls resist the pulmonary circulation and so reflect on the right heart and open the way to all the stages up to congestive failure. Close inspection of the film shows corresponding change in the pulmonary arteries which are swollen and dilated much further out from the hilum than in the normal film.

The reason is not far to seek. Such dragging out and occasional obliteration of the capillaries in the alveolar walls means that less blood is open to the residual air; there is considerable lessening

of gaseous interchange less chance for the liberation of carbon dioxide and its replacement with oxygen. At the same time there is less movement of the alveoli they cannot get the steady renewal of residual air. This is the explanation of the cyanosis of the chronic bronchitic and also of the increasing cyanosis in the bronchial asthmatic. He has the added difficulty of the muscular contraction that brings closure of the terminal lumen to the acinus. We saw the reason in discussing the muscle fibre structure of the bronchial tree. It ends in circular fashion round the last recognizable lumen at the entrance to the acinus (see Fig. 21).

The alterations on the X ray film of an advanced case are now easily visualized. The rib inter-spaces are widened. The vesicles are fewer and larger than normal so there is an increase of translucency by decrease of striations due to their walls. The pulmonary vessel markings are more in evidence. As there is less venous return the heart is smaller. The diaphragm is flattened as the blown-out vesicles press against it. We can see the cardio-phrenic angles opened out and to percussion we find much decrease in the muscle movement downwards with inspiration. The continual flattening slowly brings paresis of the muscle with consequent basal stasis of the accumulated debris.

CHAPTER XIII

BRONCHIECTASIS

BRONCHIECTASIS means a widening of the bronchial tubes. This may occur over length of the bronchus in the form called cylindrical or be localized in the form called saccula. It may be congenital in the sense that it is a complication of a prior developmental abnormality of the bronchus but is usually recognized as being frankly an acquired condition. For its production both obstruction of the lumen and infection of the weakened walls are necessary. Because of the liability of debris to accumulate by gravity and stasis in the dependent parts of the lungs we find that most cases are basal but it may appear in any part of the parenchyma as the result of blockage of its supplying bronchial lumen. Where small branchings are thus involved we have bronchiolectasis. We shall see that this is a continual accompaniment of the grosser form involving the main branches of the lower lobes.

We now recognize that bronchiectasis is not of necessity a lobar disease. Certain segments are known to be particularly affected. These are the basal segments of the lower lobes the left more than the right the lingula of the left upper lobe alone or with lower lobe basal segments and the right middle lobe. If other single segments are diseased they are almost invariably the end results of previous infections. We would therefore suspect that bronchiectasis confined to the superior segment of the left lower lobe had resulted from lung abscess in that segment.

It follows that proper treatment will depend on proper diagnosis which means full investigation of the whole bronchial tree. Uptodol is therefore injected nowadays under screen observation so that the operator can ensure filling of every broncho-pulmonary segment.

The pathogenesis is most well understood if it be envisaged as a further development of chronic bronchitis which has become ulcerative. After weakening the wall by its chronicity it has destroyed all the components of the wall from the epithelium to the muscle and elastic fibres and has finally penetrated the cartilage to infect the surrounding parenchyma.

upper sinus infection. This is a very common finding often accompanying an acute antrum trouble. Expert opinion on the upper sinuses including X ray should of course be obtained but the following points may aid in comparing the chest film



FIG. 65 The right cardio-phrenic angle in upper sinus infection.

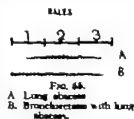
The film due to upper sinusitis usually shows much more homogeneity in its basal shadow. We do not get the same evidence of fibrosis unless the condition is of very long standing with continued reinfection of the bronchi when of course the patient is developing resultant bronchiectasis. Further the film shows a confused shadow of the inner third of the diaphragm merging into that in the cardio-phrenic angle. We do not see the lower bronchi curving inwards. We are looking at hyperemia of vessels continuing below the diaphragm.

If we look at the film of bronchiectasis at some distance from the viewing box we shall see that the striations stand out clearly as

well-defined parallel lines here and there interrupted where the cartilage is disappearing. Around them we see small opaque and hard looking shadow which are due to the collapsed and organized lobules well defined because they are offset by others among them which have complementary emphysema. That is the disease is extending by bronchiolectasis.

The next stage is perforation of the bronchial wall and consequent extension of the septa into recognizable areas of parenchyma. Its occurrence is heralded by the fluoroscope finding of râles which mean that parenchymatous material is entering the bronchus. If we listen critically we shall find they

in the middle third of the inspiration that is the bronchial parenchymatous phase. They rise in intensity to it from the first phase and then fall off in the third. They are due to air entering bronchioles that which catarrhal exudate is discharging from lobules that have been filled with that first inflammatory fluid which is the indication of irritation of the walls. In other words they are the râles of localized lung abscess.



We can recognize this exudate on the film. It is the edema of many radiological reports and is always homogeneous in its shadow having a light loss of translucency comparable with that thrown by the inferior vena cava. In this particular instance it is seen on the outer edges of a more opaque and likewise homogeneous shadow cast by lobules that have passed the stage of inflammatory exudation and are now filled with septic material. We know it connotes advancing bronchiolectasis only because we read with it the characteristic shadows of fibrosis and organization detailed above. It appears with each exacerbation of the disease later get denser as becoming true septic exudate and later still show all the non-homogeneous changes of collapse and fibrosis.

What we have now is cavity in the bronchus connecting with cavity in the lung septic bronchus continuous with septic parenchyma. To all intent and purposes we have in several years what we shall find later is the end result of chronic lung abscess where the septic lung has infected the bronchus supplying the diseased broncho-pulmonary segment. We shall find that the abscess has taken on distinct metallic emanating quality. These abscesses now present throughout two-thirds

of the inspiration over a considerable area of the lung field. Lipiodol introduced at this stage will demonstrate every step of the destructive process here we shall see only cylinders

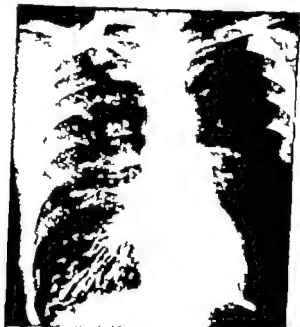


FIG. 67 Lipiodol in basal bronchiectasis.

enlargement there sacculation, at still another point nothing resembling a bronchus but only a large irregular opaque area with cotton wool edges which merge with the finer loss of transparency that indicates catarrh in irritated lobules.

Mediastinal shift will be more evident still to both film and physical findings. The fingers will show gross clubbing of the drum-stick septic type. Dullness will be much more marked and the foul sputum will contain the elastic fibres of the destroyed parenchyma.

Bronchiolectasis

We have noted how bronchiectasis multiplies itself in the minor form of bronchiolectasis by the involvement of bronchioles. Such a condition is almost always a secondary one the lumen has been blocked and then infected by septic material in the lobules

It supplies a process which copies the pathogenesis of adult tuberculosis. We shall see in the next chapter that its common cause is primary broncho-pneumonia which has not been able to get rid of its exudate. It is always a fairly localized condition and this helps us to differentiate it as a picture that has elements not dissimilar to those of broncho-pneumonic tuberculosis where the process is much more widespread into the mid zones.

The condition is not the same pathologically or radiologically as bronchiolitis condition which is discussed in the chapter on Military Shadows.

CHAPTER XIV

THE PNEUMONIAS

THE pathogeneses of pneumonia and broncho-pneumonia are fundamentally the same consisting in the interstitial reaction to the infecting organisms. In both diseases the organisms penetrate the wall of the bronchus to its surrounding connective tissue and by its lymphatics are carried to the walls of the alveoli which they first irritate, and later penetrate. In this way the pneumococci of pneumonia and the streptococci of broncho-pneumonia differ from the bacilli of influenza and Friedlander's bacilli which we have noted are carried direct to the lobules by the supplying terminal bronchioles.

The connective tissue puts up a poor resistance to the pneumococcus. In the great majority of cases the organism seems to penetrate the wall of a main bronchus such as that to the right lower lobe close to its origin. It finds no protective barrier against its invasion of the parenchyma and therefore rapidly involves the whole lobe. On the other hand the connective tissue puts up an intense resistance shown in marked inflammatory reaction to the streptococcus so that it confines the disease to patches of lobules. We can see why lobar pneumonia is a disease which spreads from the hilum in radial fashion. Its progress is rapid the invading army is too strong and sweeps aside any light opposition it may meet. It is possible however now and again to see an example where pneumonic exudate is already developed near the hilum before the first evidence of inflammatory reaction is evident in lobules nearer the periphery.

The manner of onset explains to us also the manner of clearance by either resolution or repair which must be again radial from the hilum to the periphery. The bronchi have a chance to recover from any slight reaction in their connective tissue before they are called on to deal with the parenchymal exudate which will flood them in due course to produce the typical pneumonic sputum. This manner of clearance of the disease is an important one in differential diagnosis by film examination. It is the opposite to that seen in the so-called "epituberculosis" of childhood.

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Serial film will help us to decide which we are dealing with. The picture of pneumonia is rapidly after the crisis but epituberculous will take many weeks or months to resolve and will leave on the film enlarged and crenated hilar glands and perhaps also well marked Ghon's focus at the site of the original infection towards the periphery of the affected part.

Let us try to follow the pathogenesis of developing pneumonia in more detail as we see it on the film. If there is any reaction in the connective tissue the penetration of the bronchial wall by the pneumococcus will be increased irritations along the lines of bronchial branching due to congestion by polymorphs in the peribronchial perivascular and interlobular connective tissues. These may be evident in the outer zone of the affected lobe while the inner regions are homogeneous loss of translucency. This shadow is of the same type as we found in the tension of sepsis from bronchus to parenchyma and accompanying bronchiectasis. On the outer edge we see the same fine loss of translucency due to the non-specific irritation of alveolar walls. Nevertheless the lumina have the dense shadow of true pneumonia. Update containing of polymorphs some large mononucleus and lymphocytes, in serum containing varying quantities of fibrin dependent on the connective tissue reaction. The lobules so affected fill quickly and distend and this stretching of the elastic tissue helps them to contract later so that the same elastic tissue is at the root of the bronchioles.

It is fundamental to the understanding of the physiologic effects of the X-ray film and the physical signs, to realize that when pneumonia is complicated this update replaces the residual residual remains on the last tissue of the bronchi. The alveoli are filled with fibrin and the last network of the level is therefore not stretched just as it would be in full inspiration. There is no blockage of terminal bronchioles, and therefore no collapse of lobules and no resultant shift of the mediastinum to the side of the disease. The same thing applies to uncomplicated broncho-pneumonia and uncomplicated lung abscess so that they are totally unlike in pathogenesis physiologic effects and physical signs to both bronchiectasis and adult phthisis. In these latter are proliferative diseases. The reorganization and collapse of lobules and has from their beginning associated fibrosis. They will therefore cause the mediastinum to drift towards the part of

the lung they affect so that we shall find the heart and the trachea displaced towards them

Once fully developed pneumonia outlines the lobe anatomically and the dense homogeneous shadow of its exudate remains until the crisis. This being so the shape and density in any one lobe must vary with the shape and alter with the depth of tissue in the normal lobe as we saw in our reminders on applied anatomy. The right middle lobe must be defined and dense just below the line of the lesser fissure and become more translucent towards the lower zone and unless the lower lobe is also involved we must see a clear area between the lower limits of loss of translucency and the diaphragm because this area is occupied by the lower lobe (see Fig. 25)

In the same way the shadow of lower lobe pneumonia, densest in the lower zone over the diaphragm must lessen steadily upwards towards the mid zone. It is sometimes confused with that of tuberculous pleural effusion. The physiological effects to be expected in these conditions should aid us: effusion pushes the mediastinum to the opposite side where we shall find a positive sterno-mastoid sign. If we look critically at the shadow of tuberculous pleural effusion we shall see that its peripheral density is far less than that of pneumonia and that we can usually see the ribs through it (see Figs. 5 and 16). Its loss of translucency increases steadily towards the middle of the lung field giving an impression of a band running from the axilla downwards and inwards to the diaphragm. The effusion is compressing the lung and we are looking at the area so compressed when we see this band of greater density. This is quite unlike the pneumonic density which is equal at any one level throughout its extent along a horizontal line drawn between the periphery and the mediastinum. Here the density must lessen as it goes upwards as the lobe thins out to its lingula. In other words there is less and less depth of parenchyma to be filled by the pneumonic exudate.

Moreover we shall remember the further aid to differential diagnosis. An uncomplicated pleural effusion will not blot out the vessel markings in the underlying lung but exaggerate them as there is hyperæmia but pneumonia blots out all normal striations and is usually so dense in its shadows that we cannot make out the outlines of the ribs.

The picture of fluid complicating lower lobe pneumonia can now be readily visualized for it combines both pictures. Towards

the periphery we shall see the lighter loss of translucency of the fluid and through it we can generally see the ribs quite clearly as far as the outer edge of the denser shadow cast by the pneumonia. No lung markings will appear in either shadow. Now the mediastinum will be to the opposite side whereas it was central as long as the primary pneumonia was uncomplicated. Before the fluid developed there was no sterno-mastoid sign now the muscle on the opposite side is under distinct tension.

From what we have learnt so far it follows that the slight and temporary shift sufficient to be noted by film examination in some cases of pneumonia is due either to overdistension of the lobules by their exudate or to an ephemeral blockage of their bronchioles. Close inspection of the picture should tell us which we are dealing for the effect of the former will be traction on the mediastinum without evidence of collapse the latter will present accompanying evidence of what has taken place. For example if this has occurred as a complication of right upper lobe pneumonia we shall see a distinct lower edge of the density in a sharp line. This is proof that the lingula of the lobe has shrunk upwards to the line of the lesser fissure which becomes convex upwards and is higher than its thin hair-like streak we can see about the third or fourth inter-space on the normal film. Furthermore the uncomplicated pneumonia showed normal blood vessel markings in the middle and lower lobes but now these are splashed out in order to supply the emphysematous mid and lower lobes.

To physical signs pneumonia gives us dullness due to exudate. As the lobular elastic tissue is on full stretch and maintains the inspiratory position of the bronchial walls we have no movement of the affected part. There will therefore be absence of true breath sounds but bronchial breathing will occur as the air entering the main lobar bronchus at its origin is conducted through the consolidation which acts as a sounding board.

The method of clearance of the pneumonias and the rapidity of the process is a fortunate fact for the patient. Considering all the possibilities of complication we may well marvel that we see so few cases of resulting fibrosis of the peribronchial and perivascular tissues so recently recovered from their initial reaction to the invading organism have now to deal with lymphocytes and plasma cells. These again produce irritations on the film and they can remain for a considerable time. They should not lead us to give bad prognosis by reading them as evidence,

fibrosis unless we find they are accompanied by the characteristic crepitations of this condition. But they can produce fibrosis as we shall discuss in more detail later.

The fibrinous exudate in the lobules is coughed up or absorbed after being liquefied by a proteolytic ferment supplied by its accompanying polymorphs. It is because of this liquefaction that we hear the so-called *redup* crepitations of pneumonia. If we listen to them critically we shall find that they are really sticky sounds at the very end of inspiration and are rales rather than crepitations. We are hearing air entering the alveoli through the exudate which is now discharging into terminal bronchioles.

It may be that poor supply of polymorphs in the exudate is the basic cause in those cases that go on to fibrosis. Capillaries have time to enter it before it is liquefied. In any case we do know that fibroblasts replace the fibrin passing through the alveolar walls and from lobule to lobule causing organization of the exudate that remains and consequent shrinkage collapse. Although others around these lobules are bound to show some complementary emphysema there will be shift of the mediastinum to the affected side. Normal unaffected tissue is being dragged over by the intrapleural pressure. Chronic interstitial pneumonia as it is called is therefore exaggerated repair tissue. It is usually found in one lung and we can see how it differs from the fibrosis of chronic bronchitis due to bilateral long-standing hyperæmia because this latter is a peri-bronchial tissue increase not an interlobular one. If it is very severe it can crush the supplying bronchioles and give us scattered areas of lobular collapse which is not an unusual finding.

It follows that the X-ray of chronic interstitial pneumonia will show the ribs on the affected side closer together than normal and that the diaphragm will be higher than normal while the heart and mediastinum will have drifted towards the fibrosed lung. Throughout the diseased lung will be strands of fibrosis which have no distribution corresponding with that of the normal blood supply. These will be more prominent in distant viewing and will be seen to have scattered among them nodular like shadows which are cast by organized bunches of alveoli.

Auscultation will give us widespread crepitations which reach their loudest point towards the end of inspiration as they are parenchymatous in origin.

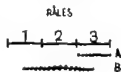


FIG. 68.

A Pneumonia.

B Broncho-pneumonia.

This condition is quite unlike the results of the pneumonia of the new born because here at lectasia is preceding entity the lobules never opened at all which is necessarily a different condition from acquired collapse. It is also quite unlike so-called post-operative pneumonia which is not pneumonia, but a purely bronchial disease. A sudden thick sticky exudate from the wall blocks the lumen with the same dramatic effect seen with an inhaled foreign body. Residual air cannot be renewed, the parenchyma collapses and there is no obstacle to the action of the intra pleural pressure on a lobe that perforce must hink to its main bronchus at the hilum. Screening the patient or taking two films, one on complete inspiration and the other on complete expiration, will show us what never happens in ordinary pneumococcal pneumonia. Winging of the collapsed lobe, the heart and the mediastinum with each inspiration to the affected side. There will be some elevation with each expiration but still displaced mediastinum which will be shown by the positive medio-mastoid sign even if it be impossible to demonstrate by the position of the apex beat. If the glutinous material is not cleared out by postural drainage or bronchoscopic suction the consequences can be very serious. Organization can lead to every stage of bronchiectasis the same end result so often seen in the pneumonia of the new born. To this last condition the name congenital bronchiectasis may be applied with us at lectasia present from birth there is often maldevelopment of bronchi so that cysts are frequently found and these the superimposed infection turns intoumps of septic material to add to the bronchiectatic end result.

From what has been said of the pathogenesis of pneumonia the possibility of localized forms can be easily envisaged. The organism travels some way along the main bronchus before penetrating the wall of one branch. This is comparatively common in the pneumonia of childhood which gives rise of its forms the so-called hilar pneumonia by involving the posterior horizontal branch of the right lower lobe bronchus. It is not uncommon to find in an adult that only the pectoral or the axillary branch of the right upper lobe bronchus has been affected so that we see on the film that triangular shadow of the new parenchyma already referred to in the section on applied anatomy (see figs 14 and 15). The development of the disease is just the same as in lobular pneumonia. It spreads outwards from the point at which the

bacilli penetrated the bronchial wall so that all the segment of lung supplied by that bronchus is involved

The shadow cast by these localized pneumonias is never really like that of tuberclosis with which it is usually considered in differential diagnosis. Both may give a triangular loss of translucency in the outer axillary region of the right upper lobe the pneumonic one being due to involvement of the axillary branch of the main bronchus while the tuberculous one is due to a tuberculous deposit in the periphery of the outer base of the upper lobe that is commonly just above the peripheral end of the lesser fissure. Both cause thickening of the fissure. Both can show abnormal films for a long time after onset because localized pneumonia tends to heal by repair rather than by resolution. If however we look critically at serial films we shall find that the tuberculous shadow always has its main continuing density against the periphery as it is essentially a parenchymatous disease the bronchi are involved only secondarily at all stages. This is not so



FIG. 69 Recovering partial pneumonitis of right upper lobe

with the localized pneumonia healing by repair the main density is now at the apex of the triangle that is round the supplying bronchus at its entrance. Secondary involvement of bronchi in tubercle is at the site of the deposit; there the bronchi dilate steadily until, in most cases, definite cavitation is seen.

Another shadow that may take considerable time to clear after pneumonia is that of the exudate on the pleura especially if the interlobar pleura has been affected. In all cases there is a true

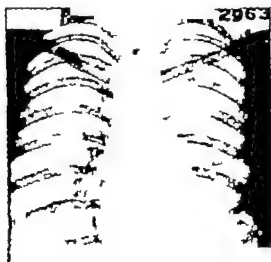


FIG. 70. Filling of left costo-phrenic angle in recovering pneumonia.

pneumonic exudation on the visceral layer and this causes adhesion to the inflamed parietal layer. Movement by inspiration therefore causes sharp stabbing pain. Liquefaction usually leads to rapid absorption but we have already noted how absorption may take long time in the costo-phrenic angle and in the interlobar fissure so that we get filling of the costo-phrenic angles and tenting of the diaphragm that are permanent but of no clinical import. In the

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same way the lesser fissure may be thickened for a considerable period after a partial pneumonia.



FIG. 71. Lateral view of fluid in major fissure producing "wedgeing."

Broncho-pneumonia

Broncho-pneumonia as already stated may be considered as consisting of localized patches of pneumonia confined by the strong resistance of the peribronchial connective tissue which limits the disease to bunches of lobules. It is commonly bilateral from its onset, but it may be completely unilateral, or develop on one side later than the other. It is usually in the lower lobes but is not uncommon in the right middle lobe.

Its progress can be followed on the postero-anterior film, which demonstrates shadow that begin near the cardiac border and increase in size and in spread along the bronchial distribution so that they go downwards and outwards in lower lobe involvement and almost horizontally outwards in middle lobe disease. The involved areas show the same homogeneous losses of translucency as we saw in pneumonia only now they are in the greater densities triangular in shape.



FIG. 72. Broncho-pneumonia, right middle lobe.

particularly in the middle lobe as the lobules are pictured along the side of their cone-shaped broncho-pulmonary segments. The



FIG. 73. "Contact" Shadow of right lower lobe as in Fig. 54.

lobules involved by pneumonic exudate are never entirely discrete because they have around them those showing the first inflammatory non-specific catarrhal change. There is no shift of the mediastinum.

The picture is therefore in two characteristic unlike that of broncho-pneumonic phthisis. Firstly this disease is fibro-carcinoma and will show local areas of actual collapse among others with emphysema complemented by emphysema. Despite of the fact that both conditions may be surrounded by edematous shadow. This will be quite evident if we look at the films at some distance from the viewing box when the shadow of streptococcal broncho-pneumonia will be no more distinct but those of tuberculous broncho-pneumonia will become much more clear. Secondly there will always be some collapse and therefore some displacement of the mediastinum with phthisis and if this is not taken to X-ray it will be to physical examination.

The physical findings can be explained by the pathogenesis and the X-ray picture. We shall find putty areas of consolidation lobules filled with distension with pneumonic exudate and innervated. The disease itself is developed we shall get grip of this exudate entering the terminal bronchioles. The lobe involved is never

of course completely silent there are parts which have escaped altogether and others where only inflammatory exudate occupies lobules still maintaining some airway. If we listen carefully we hear rather bubbly rales in the mid third of inspiration because this exudate is discharging into terminal bronchioles (see Fig. 68). This is a distinctively stethoscopic finding throughout the clinical illness the only difference being that the rales get more marked as the pneumonic exudate liquefies.

The dullness will naturally be much less than that of pneumonia as the involvement is patchy but there will be distinct lack of movement laterally in cases where the lower lobes are diseased.

By reason of the much more intense connective tissue reaction many cases proceed to small areas of atelectasis. The increased tissue naturally gathers round the terminal bronchioles of the affected lobules. This interferes with the natural movements of the bronchioles in response to respiratory action; that is their muscle fibre action is inhibited. Their elastic tissue reaction which is dependent on the stretching of the alveoli to residual air is already interfered with, since the alveoli are fixed at full expansion by the exudate in them. When the disease tries to resolve these bronchioles are therefore caught at a distinct disadvantage. They have no time to recover from this initial irritation of their surrounding connective tissue before they are flooded by lymphocytes and plasma cells from the exudate now trying to escape from the alveoli as resolution sets in. We have now irritation of the inner wall of the bronchioles added to their connective tissue irritation on their outer walls. They are therefore all too easily blocked so that alveoli that have managed to empty go on to collapse while those still containing exudate go on to organization.

We can see therefore the inherent danger of too early movement, perhaps advised by the belief that modern methods of treatment by reducing temperature have brought resolution. If this is dangerous in lobar pneumonia it is much more dangerous in broncho-pneumonia. If the bronchioles do not empty their walls weaken and the way is open to bronchiolectasis and later to bronchiectasis.

This is especially possible in right middle lobe disease. We have already noted that its main bronchus goes out almost horizontally from the right main bronchus. It is therefore difficult to drain. If it goes on to blockage in its terminal bronchioles we have a slowly changing picture and accompanying changes in

physical sign. The areas that on previous distant viewing merged with the surrounding catarrhal inflammatory exudate now stand out with the clarity of broncho-pneumonic phthysis with which we may confuse them because of the accompanying shift of the mediastinum. We shall not however forget that they are confined to lateral densities in one area of the lower mid zone as against the widespread distribution of tuberculous broncho-pneumonia and that they have by much more exaggerated peribronchial fibrosis. A lateral film would show us how much more the lesser fissure shares in this fibrosis than it does in phthysis and of course, the general condition and the lower and less-rising temperature will be a further aid. Both conditions will of course give us the crepitations of fibrosis but they will be much more localized and definitely coarse and more insistent in complicated streptococcal broncho-pneumonia. Many cases seem to go on to simple fibrosis in the lower mid zone. Indeed the fortunate patient may escape further clinical upset but many go on to slowly increasing bronchiolectasis and the established and progressive bronchiectasis. This is a common end-result long afterwards by superimposed infections.

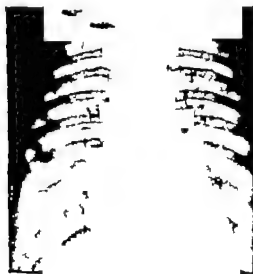


FIG. 74. Fibrosis following right middle lobe broncho-pneumonia.

The patient may for a long time be unaware of what has happened. Mass radiography has shown that many apparently completely



Fig. 75. Lordotic view of same patient as Fig. 74.

recovered cases of middle lobe broncho pneumonia have been left with much fibrosis. The X rays shown in Figs. 74 and 75 are an example. The first demonstrates a loss of translucency in the right lower mid zone. The patient had signs of fibrosis in this area. The lordotic picture shows how the middle lobe has shrunk very considerably. Lipiodol later proved the presence of bronchiectasis.

An important point to remember is that every case of broncho-pneumonia causes marked increase in Hila shadow because of the intense hyperemia of its resistance; the hilar glands also swell with edema. This hilar increase takes a long time to clear up and this is especially noted with the usual type of scoliosis which brings the left hilum into prominence. There is the possibility of a grave diagnosis of localized glandular disease but the swelling is seldom of the extent of Hodgkin's disease and the swing of the Peltz-Ehrlich fever and the blood count will be further aids in doubtful cases. The enlargement remains long after the patient is clinically well.

CHAPTER XV

ATELECTASIS

In dealing with this condition which may be defined as collapse of parenchyma through loss of residual air we shall confine our discussion to the acquired form due to obstruction of the lumen supplying a broncho-pulmonary area. This may occur by something inside the bronchus such as a growth or inhaled foreign body blood mucus or granulation tissue or by something pressing on the wall of the bronchus such as an enlarged gland or tumor formation. As we saw in our studies of applied anatomy and in the description of post broncho-pneumonic atelectasis in the right

liddle lobe the size of the affected cone depends on the size of the bronchial lumen. The same we shall find to be true in the secondary results of blockage of the terminal respiratory bronchiole in adult phthisis where granulation tissue enters it from the primary acina deposit. If main bronchus is occluded, as in post operative pneumonia then the whole lobe collapses since the residual air to every terminal alveolus is absorbed.

In such condition oxygen is first absorbed by the capillaries in the alveolar walls and we get on the film fine loss of translucency. We cannot see this taking place in the small atelectatic areas of bronchiectasis or complicated broncho-pneumonia but we can follow it quite easily where a whole lobe is shutting down, and we shall in fact find this light homogeneous shadow beginning in the least supported areas of parenchyma. Thus in the upper lobe it will occur first in the outer apex and in the lower lobe along the antero-basal margin; we saw the reason in discussing structural support under our considerations of applied anatomy these areas are dependent almost completely for their normal position on the residual air and have but little support from their scaffolding of connective tissue. Later the nitrogen is absorbed and the shadow while still homogeneous becomes denser by more dense

Up to the time when such density covers the whole of the affected lobe it may be possible in those cases that are due to carcinoma of the bronchus to differentiate on the film the homogeneity of

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FIG. 75. Lordotic view of same patient as FIG. 74.

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In such a condition oxygen is first absorbed by the capillaries in the alveolar walls and we get on the film a fine loss of translucency. We cannot see this taking place in the small atelectatic areas of bronchiectasis or complicated broncho-pneumonia, but we can follow it quite easily where a whole lobe is shutting down and we shall always find this light homogeneous shadow beginning in the least supported areas of parenchyma. Thus in the upper lobe it will occur first in the outer apex and in the lower lobe along the antero-basal margin. We saw the reason in discussing structural support under our consideration of applied anatomy these areas are dependent almost completely for their normal position on their residual air and have but little support from the scaffolding of connective tissue. Later the nitrogen is absorbed and the shadow while still homogeneous becomes definitely more dense.

Up to the time when such density covers the whole of the affected lobe it may be possible in those cases that are due to carcinoma of the bronchus to differentiate on the film the heavy opacity of

the actual growth near the hilum, next to it the slightly less opacity of those areas already completely collapsed and towards the periphery the finer loss of translucency of early collapse. We have here a characteristic 'whitewash brush' film. It looks as if the brush had been applied heavily over the point of entry of the bronchus and then as it was swept outwards towards the periphery had been pressed with continuing less force. The apex of the

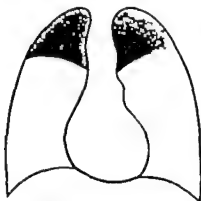


FIG. 74. Carcinoma left upper lobe bronchus, compared with tuberculous pneumonia of right upper lobe.

broncho-pulmonary area is densest its base least dense. Later as the lobe becomes smaller by shrinkage and is followed up by its proximal lobe which develops *pari passu* its complementary emphysema the interlobar fissure rises and the density becomes throughout equal to that of the causative growth, which indeed in most cases has by this time extended by its cells, or its mucinous exudate into a considerable portion of the surrounding parenchyma.

In many there appears by now the further complication of abscess bringing a more or less central area of translucency.

From the beginning there will be lack of movement and dullness will deepen with increasing involvement of the growth. The mediastinum will drift to the same side. We shall find the evidence of shrinkage in alteration of the normal lesser fissure or the appearance of the greater fissure on the postero-anterior film and in the change in blood supply striations in those parts of the lung that are blown out by complementary emphysema. Lateral films are of particular assistance as we have already noted in all conditions effecting change in size and shape of lobes.

It follows that the dullness of collapse must be accompanied by the tympany of emphysema. This is why broncho-pneumonia and its complication of terminal bronchiole blockage never give over all dullness and why with the dead dullness of the collapsed right upper lobe we find marked tympany in the compensatory distension of the middle and lower lobes.

Bronchial breathing may be very marked just short of the actual growth at the origin of the affected bronchus. Added sounds inside the collapsed lobe do not present invariably in the



FIG. 77. Carcinoma right upper lobe.

upper lobes unless there is damming back of secretion and the complication of abscess is superimposed. Once the extension of growth begins to press on the bronchus to a lower lobe we get, beyond this second bronchus, a very high pitched musical wheeze from air passing the obstruction, as in the production of an instrumental whistle. Its appearance is of grave import.

The most constant sign of collapse in all its forms is the sternal-tenderness sign, which is always positive on the affected side. In the difficulties of differential diagnosis it is a much more reliable finding than any other single sign, even including comparison of films on complete inspiration and complete expiration. We have already noted how the trachea reacts to changes in normal lung elasticity, the shrinkage of the affected part leaving the remaining normal tissue open to both increased pull on the same side and increased push from the other. While this tracheal reaction is

constant the position of the heart is variable for as we saw its greatest weight is in the lower mediastinum, and it will react only to lower zone disease or complete unilateral disease. Even then it is unwilling to respond to anything minor in force. It will swing on its long axis and can be pushed backwards by the emphysema of the opposite side before it has moved bodily and laterally to the affected side. While it must certainly always be located by palpation and by stethoscope the position of the apex beat in collapse may be a misleading sign. Even to screen examination the heart may show no definite shift with inspiration if the diseased area is above the origin of the aorta. These tests we shall see applied in more detail in considering atelectasis of individual lobes.

Upper Lobe Atelectasis

The commonest cause is carcinoma of the bronchus. It is here we see best demonstrated the "whitewash brush" effect on the film although the very first evidence of interference with residual air may appear in a fine loss of translucency in the outer infraclavicular region. Quite early we can see the accessory signs in shift of the trachea and the mediastinum to the affected side and in the upward shrinkage of the interlobar fissure on to which the thin triangular lingula retreats steadily. The interlobar fissure becomes more and more evident because the rays are passing through more and more tissue. It has added to it all the tissue of the retracting lingula of the lobe. These findings are always more easily recognized on the right as against the left side as the shadow of the upper right cardiac border is made in great part by the movable superior vena cava, usually less well confined than other mediastinal structures by the surrounding areolar tissue. The heart does not move much in the lower two-thirds of its extent and so there may be no appreciable change in the position of the apex beat to palpation or auscultation even with considerable shrinkage of the upper lobe.

It is quite possible that in its early stages the film can resemble that of a developing pneumococcal pneumonia although temperature symptoms and the examination of serial films should help us to avoid misinterpretation, and we know that an uncomplicated pneumonia does not produce mediastinal shift. A lateral film will aid us because we shall see the retraction of the lobe on to the inner end of the lesser fissure in a manner never displayed with developed pneumonia. Further we shall find that while pneumonia gives

dullness both anteriorly and posteriorly that of collapse is always anterior and below the lower end of the collar bone and the left of right of the bronchus.

The difference in site of dullness is of distinct help if the diagnosis is against upper lobe pneumonia. Phthisis here the dullness is posterior as tubercle in adult form is posterior disease inclined to attack parenchyma supplied by distally inclined branches of the bronchial tree. Again tuberculosis is parenchymatous disease in its pathogenesis not bronchial disease and the greatest density in its picture is noted below the middle third of the collar bone and not the left of the entrance of the main bronchus.

Right Middle Lobe Atelectasis

We noted when dealing with right middle lobe broncho-pneumonia that the dangers of inefficient bronchial blockage are considerable owing to the angle it makes with the main lobar bronchus.

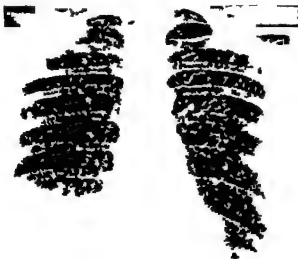


FIG. 78. Collapsed right mid-lobe from broncho-pneumonia.

comes off the right bifurcation. Post-pneumonic flaccid by acid sputum is very common and occurs in many cases with perhaps little immediate but grave after-effects. It can come on quite

suddenly or very slowly. In the latter case the pain has a characteristic position along a band below the right nipple coming in waves of intensity up to a pitch of severe discomfort.

The postero-anterior film gives a shadow outlining the lobe, heaviest along the line of the lesser fissure and lessening to an area of translucency above the diaphragm in spite of the fact that



FIG. 79. Collapse right middle lobe, by innocent intra-bronchial growth.

this muscle rises towards its position of expiration against the emphysema of the lower lobe. The lateral film is characteristic and unlike that of any other disease. It is almost always of the whitewash brush type dense at the entrance of the bronchus and fading towards its base. The upper side of the triangle is along a line beginning at the junction of the middle and lower thirds of the mediastinum and going horizontally outwards to the posterior aspect of the sternum. The inner side of the triangle goes downwards and outwards to a point near the middle of the diaphragm. The case demonstrated in Fig. 79 was one of collapse due to

benign tumour of the middle lobe bronchus the growth was removed through a bronchoscope



FIG. 80. Lateral view same case as in Fig. 79

The lateral film of collapsed middle lobe can cause difficulty in diagnosis as against effusion in the lower fissure. The upper



FIG. 81. Lateral view in pleural effusion

border of the fluid is seldom straight as in collapse. It is inclined to bulge upwards in concave fashion and even if it is convex downwards because the fluid is not even layered, we do not get evidence of emphysema in other parts of the lung. Physical signs should also help. The fluid will be encysted, dense and restricted in area before it causes drift of the mediastinum to the side and it will not give us the marked tympany of complementary emphysema which is bound to accompany collapse.

The swing of the heart to the affected side by inspiration is more marked in middle lobe than that found in upper lobe collapse. The two characteristics to physical examination are the positive sterno-mastoid sign and dullness which is anterior in the lower mid zone. The emphysematous lower lobe gives definite posterior tympany.



FIG. 82. Fluid in left interlobar fissure.

The only other condition giving such marked anterior dullness is fluid in the main interlobar fissure but its films antero-posterior, lordotic and lateral are entirely different, the first showing a low

of translucency sitting below the diaphragm like that of pleurisy secondary to sub-phrenic abscess, the lordotic tent like shadow with its base on the diaphragm and the lateral density between the cardiac shadow and the diaphragm.

As already noted many cases of post broncho-pneumonic collapse of the middle lobe can pass unrecognized at the time of the clinical illness as there is much less upset in pulse and temperature than occurs in the average case of lower lobe post-pneumonic collapse. Only considerably later after what then appears to have been a rather protracted convalescence does the patient complain of dragging pain; full investigation including lipiodol injection, commonly shows considerable dilatation of the bronch. Bronchiectasis is by no means rare sometimes by superimposed infection but often only by the continuing effects of the collapse which has led to tissue formation by the damming back of the exudate.



FIG. 83. Pleurisy secondary to abdominal disease.

This type of case often presents one of the most difficult problems of differential diagnosis because the abscess almost always produces the complication of a frank pleurisy with the danger of consequent empyema. We are now presented with a condition which is all too easily read as pleural effusion. Fortunately the tracheal displacement in response to atelectasis is always more marked than the pressure of the fluid in the wards the tympanic sign remains positive on the right. The writer has seen several cases where this was the main indication to the true diagnosis. It is of course taken for granted that all other clinical aids including needling will be employed for every ultimate diagnosis.

Lower Lobe Atelectasis

The commonest types result from pneumonia, broncho-pneumonia, post-operative pneumonia and new growth of the bronchus. Of these the most sudden in onset and the most massive in result is the post-operative due as we have already noted to the formation of a glutinous exudate on the wall of the main bronchus. The pneumonic and broncho-pneumonic types

border of the fluid is seldom straight as in collapse. It is inclined to bulge upwards in convex fashion and even if it is convex downwards because the fluid is now encysted we do not get evidence of emphysema in other parts of the lung. Physical signs should also help. The fluid will be encysted, dense and restricted in area before it causes drift of the mediastinum towards it and it will not give us the marked tympany of complementary emphysema which is bound to accompany collapse.

The swing of the heart to the affected side by inspiration is more marked in middle lobe than that found in upper lobe collapse. The two characteristics to physical examination are the positive sterno-mastoid sign and dullness which is anterior in the lower mid zone. The emphysematous lower lobe gives definite posterior tympany.



FIG. 82. Fluid in left interlobar fissure.

The only other condition giving such marked anterior dullness is fluid in the main interlobar fissure but its films antero-posterior, lordotic and lateral are entirely different, the first showing a loss

this has less structural support, as well necessarily as less residual air for absorption. The blood vessels of the partially collapsed lobe are crowded towards the mediastinum which is displaced to the side of the lesion. With this the most marked change in physical signs is the sharp tension of the sterno-mastoid on the same side. It was unaffected by uncomplicated pneumonia. At the same time we note that the dullness which had been clearing both anteriorly and posteriorly is now again marked posteriorly this is because the lung shrinks downwards and backwards as well as inwards. The last two movements tend to make the heart swing on its axis: thus in right lower lobe collapse the right lower border recedes and the left lower border comes forwards and to the right. The apex beat may therefore be but little disturbed from the normal, and is not a reliable aid to the diagnosis. The swing of the heart on inspiration to the affected side will be quite evident to screen examination.

In the second stage the shadow is much more dense. Its costal border goes downwards and outwards towards the mid-point of the diaphragm on the affected side. So dense is the shadow that it appears to be continuous with and equal to the heaviest density of the heart shadow towards its mid-point. There is therefore total obliteration of the cardio-phrenic angle. It follows that this shadow ought not to be confused with that of a squat heart. In cases of left lower lobe collapse the diaphragm is higher than normal in both conditions due to lobar shrinkage in collapse and to its being pushed up by abdominal fat in the case of the squat heart but the shadow is essentially different.



FIG. 85. Lower lobe atelectasis, Stage II.

Moreover there are further film and physical findings which are conclusive. Collapse as we noted must bring emphysema. In this case it is in the costo-phrenic angle, where we can see marked loss of normal lung markings on the film while in the upper and mid zones we find the vessel-markings splayed out much more radially than in the normal. There will therefore be marked tympany in the axilla, off-setting the increasing dullness in the lower inner third posteriorly and we shall hear the blowing breath sounds from the bronchus over its point of entry near the

are usually slower in development and therefore approximate to the slower shrinkage seen in bronchial carcinoma.

The characteristic shadow of the preceding pneumonia has been described and the changes in broncho-pneumonia which goes on to lobular and lobar collapse have been detailed. In pneumonia there has usually been considerable progress towards recovery with clearance of the homogeneous density from the crisis in temperature before the complication occurs. When collapse sets in by blockage of the main bronchus by a plug of sputum the film goes through a series of alterations similar to those seen in both post-operative pneumonia and bronchial carcinoma that is density begins again at the hilum.

It is essential to remember that the same picture can result from abscess and from tuberculosis at the apex of the lobe (see Fig. 17). Radiography can fail altogether to give the true cause of what is seen. It may have been possible to follow the developing collapse from a rounded opacity close to the hilum but usually one sees only a partial collapse in the first X ray the apex of the lung being already hidden in the hilar shadow. Full clinical and bacteriological investigation and serial X rays must be considered in every case.

Three stages of lower lobe collapse may be detailed: early, half and complete collapse. All three have the homogeneous type of shadow that is it may alter in density but not in smoothness throughout its extent, no part of it but only the whole standing out on distant viewing as the concurrent emphysema is outside the boundary of the affected lobe. All three stages have the same upper point to their loss of translucency. This lies at the upper limit of the ventricular or auricular shadow on the cardiac border. Left and right sided cases are similar in this apical hilar limit to their triangular shape.



FIG. 84 Lower lobe atelectasis, Stage I.

The first stage shows a loss of translucency diminishing from this point downwards and outwards to the periphery the upper border being made by the interlobar fissure. It will be realized from the anatomy of the lobe that this means that its apex has already undergone a considerable fall through shrinkage of the triangular lingula, and this we would expect as the lingula being

The third stage is shown on the film as very dense area heavier than that of the cardiac border rather like a collapsing ton balloon with its neck attached to the upper cardiac border. On the right side its outer edge is usually concurrent with the right cardiac border on the postero-anterior film while on the left side it lies within it. It is in such cases in particular that the complementary emphysema may be the only indication of abnormality. By this stage there is emphysema on the opposite as well as on the affected side and this has an important bearing on physical findings. We have learnt so far that the mediastinum is one continuous whole not a structure with



FIG. 32. Lower lobe atelectasis, Stage III.

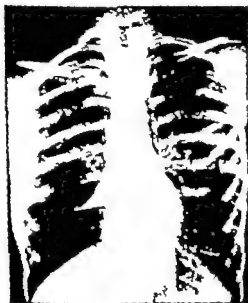


FIG. 33. The mediastinum and heart have no substance.

eighth or ninth dorsal vertebra because the collapsed lung is acting as a sounding board



FIG. 86 Right lower lobe collapse to second space.



FIG. 87 Squared heart.

this will undo the tendency to swing. It has brought the sub-atmospheric pressure nearer to atmospheric or above it. The muscle tension still remains the pull on the trachea by the uncollapsed parenchyma is still present. If therefore we find a patient who is dull all over the lower half of his chest both front and back and in the axilla, on the left side and still has a tense left tympanic drum he cannot have uncomplicated pneumonia because pneumonia does not move the mediastinum equally he cannot have left lower lobe pneumonia with pleural effusion because this would give a right sterno-mastoid sign and so he must have collapsed left lower lobe with pleural effusion.

right and left sides. As one side of the thorax shrinks, therefore the mediastinum drifts as a whole. In collapse of the right lower lobe with drift of the mediastinum to the right there is also drift to the right of the left lower lobe which by emphysema attempts to fill the vacated space. This it does by occupying the lower anterior mediastinum and in doing so it must perforce overlap the anterior surface of the heart pushing it back wards. We have therefore seen two reasons why the heart swings and goes backwards rather than move to the side of the atelectasis during conditions of respiratory rest. Herein lies the reason why we get with complete collapse anterior dullness in the affected cardio-phrenic angle—we are percussing the heart pressed back to be continuous in note with the much shrunk lower lobe. All over the remainder of the lung and particularly in the lower axillary zone we shall find marked tympany.

If however we screen the patient or take two pictures—one on complete inspiration and the second on complete expiration we shall see the effect of the strengthened intra-pleural pull on the affected side for the heart comes well over with each inspiration. This finding combined with a tense sterno-mastoid muscle will aid us in diagnosis but we shall find the latter the better help if the patient has the complication of fluid in the pleural cavity for



FIG. 90. Left lower lobe collapse with fluid—sterno-mastoid sign positive on left side.

ATELECTASIS

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this will undo the tendency to swing. It has brought the sub-atmospheric pressure nearer to atmospheric pressure above it. The muscle tension still remains the pull on the trachea by the uncollapsed parenchyma is still present. If the effusion be found a patient who is dull all over the lower half of his chest both front and back and in the axilla on the left side and still has a tense left sternal mastoid he cannot have uncomplicated pneumonia because pneumonia does not move the mediastinum equally he cannot have left lower lobe pneumonia with pleural effusion because this would give a right sterno-mastoid sign and so he must have a collapsed left lower lobe with pleural effusion.

for the same reason as it is not of equal density in its rounded area of loss of translucency on the film. It is heaviest in its centre as here are the lobules nearest the bronchial branch along which came the aspirated material. Outside these are the lobules which have been irritated in their alveolar walls so that they contain catarrhal inflammatory exudate giving the fine loss of translucency of oedema. The homogeneity of shadow is a diagnostic X ray feature and we shall see it remains until cavity formation occurs in those cases that go on to blockage of the bronchus. We see therefore that it is of the same consistency as the shadow of lobar pneumonia and of the individual shadows of broncho-pneumonia all three having in common material which is filling lobules and replacing their residual air. Putting this in another form, the previous lobar shadow of pneumonia is now in a more or less circular localized area the shadows of broncho-pneumonia are now confluent in a larger single area.

Sometimes abscess involves a considerable area showing no special denser central region but only a wide-spread area of homogeneous loss of translucency. There is no particular definition such as appears in lobar pneumonia, making out a definite lung segment.



FIG. 92. Lung abscess.

At first the area must be silent apart from its complicating pleural rub. It will cause no shift of the mediastinum as its material has the same physiological effect on lobular latic tissue as the residual air it replaces. There will be no terno-mastoid sign nor will it be found in the usual following stage of the abscess which evacuates voluntarily. This stage is often referred to as a cavitation because the picture on the film is misread there is no real difference except in extent between this second stage and the second evacuating stage of a broncho-pneumonic single lobular affection. Naturally the lobules nearest the draining bronchus evacuate first so they make central translucency within a circular homogeneous shadow made up of those lobules still undrained. This ring shadow has no components of organization or collapse. It does not alter within itself or stand out against any surrounding emphysema with distant viewing as a tuberculous cavity wall does (see Fig. 48). Stethoscopic findings bear this out as bear just the same files as we heard in broncho-pneumonia in the mid-phase of inspiration indicating lung material entering the bronchus but naturally they are increased in number and coarse new as a bigger circumscribed area is affected.

It is necessary to repeat again even if it appears redundant that the usual lung abscess is not cavity as many students seem to visualize the process. It is merely to all intents and purposes a very localized pneumonia filling lobules with exudate as pneumonia does and differing only by the method whereby it was caused in these lobules. It distends them as pneumonic exudate did and does not destroy them. Most patients evacuate the material voluntarily at a stage when it contains no latic fibres. Moreover this method of cure never has with it any lubbing of the fingers. Should however this evacuating material block the bronchus the septic material has not been dammed back.

Should however this evacuating material block the bronchus the way is open to serious consequences from cavitation to bronchiectasis. The first hint can often be got by the film examination the exudate now fluid and thin by the proteolytic enzyme of its polymorphs is dammed back and we see level within the diseased area, confined by the circular real hape of outlying lobules which are still filled with large mononuclear phagocytes and polymorphs. Should this not be evacuated by drainage by conservational or surgical methods then repair and not resolution will take place in these surrounding lobules. They cannot find an

exit for their material content and slowly fibroblasts will enter them organization and collapse will follow drag on the bronchus be the inevitable result and bronchiectatic cavitation by advancing sepsis from lobules to bronchus is the final picture The septic material now destroys the parenchyma, and the foul sputum contains elastic fibres There will be an immediate reaction in the mediastinum To all intents and purposes we have one single cavity of bronchiectasis There we saw how sepsis in the bronchus extended through all the coats of the bronchial wall until it reached the parenchyma and produced a cavity in the lung connected with a saccular cavitation of the bronchus This is just the reverse process leading to the same end result The disease can now extend like any individual cavity in primary bronchiectasis

The physical signs are now those of organization fibrosis and atelectatic collapse By destruction of elastic tissue we have drift of the mediastinum with a positive sterno-mastoid finding to the stethoscope we have the crepitations of fibrosis and the coarse metallic riles of bronchial dilatation and its connection with lung tissue are heard throughout the first and mid phases of inspiration These are exactly the same added sounds as we found in advancing bronchiectasis

Clubbing of the fingers of the septic type comes on rapidly with the blockage of the bronchus If evacuation by efficient drainage is carried out the clubbing disappears quite soon; if it does not do so then the observer should be doubtful if the lung condition is really cured Many cases are shown by tomography to be going steadily if slowly forward to the stage of bronchiectasis in spite of apparent cure by operation This is why there are so many so-called recurrences of lung abscess they are not recurrences but continuances of the same initial uncured lesion We are now dealing with secondary bronchiectasis not a lung abscess

CHAPTER XVII

TUBERCULOSIS UP TO EARLY ADOLESCENCE

Whatever be its out of entry to the body by inhalation the tubercle bacillus can always reach the lungs. When it comes in contact with any mucous surface it is ingested by a phagocyt carried by it through the mucous membrane to enter the lymph spaces and thence is conveyed to the lymphatic node or tissue draining this particular area. Its next stage on the journey is the venous blood and so the right heart is reached and, through the pulmonary circulation the lung tissue. Should it be held up in a small capillary the phagocyte pierces the wall to enter the surrounding lymphoid tissue and then it forms a focus of disease. In other words whatever be the manifestation of the disease in the lungs from childhood to old age tuberculosis is primarily lymphatic disease. Its forms depend on the reaction of lymphoid tissue. It always attempts to drain along the lymph channels which we saw in our consideration of applied anatomy are part of the protective system together with the lymph spaces and the glands. It succeeds in most cases in the infections of childhood type but its very evidence of acquired immunity in its efforts to confine the infection to localized areas can be the undoing of the infected subject as age advances. Since the lymphoid tissue, so hedged about becomes the breeding-ground for the bacilli and so itself breaks down. It is worthy of note that increasing age brings much increase in lymphoid tissue throughout the lungs thus is doubtless in great part an explanation of the widespread ravages of senile phthisis which yet maintains such resistance as to limit only chronic bronchitis.

Although there are no sharp lines of demarcation dividing the disease into forms of different ages it is convenient to describe its further developments under the headings of Infancy and Early Childhood, Childhood to Early Adolescence, and Early Adolescence and Adult Life as they show fairly defined types of response of the now infected organism to fresh infections.

Tuberculosis in Infancy and Early Childhood

The forms of the disease shown in this age-group are miliary

CHAPTER XVIII

ADOLESCENT AND ADULT TUBERCULOSIS

EMITUBERCULOUS lesions are found up to about the fifteenth year of life although most of them occur before the age of twelve. Therafter we come to the manifestations of tuberculosis most commonly met with in general practice. Fundamentally they are the same as those of earlier ages in their interplay of infection and immunity that is they are probably due in the most part to reinfections or reawakenings of previous primary infections. It is highly possible however that many of them are continuing primary infections in individuals who have not been infected before the age of adolescence. Some patients show exudation with no foregoing or with destroyed foregoing immunity others show lessened immunity and thus varying amounts of consequent fibrosis. Some therefore show the spread of the blood-borne type of miliary tuberculosis some show this form combined with fibrosis in the individual scattered lesions of chronic miliary tuberculosis. While others show break into a bronchus and give broncho-pneumonic or pneumonic types. Most however show the disease commencing as far as we know by the diagnosis through X ray films in those special broncho-pulmonary segments of dorsal branches of bronchi which we studied under applied anatomy and saw involved in the commoner sites for formation of lung abscess. Considerable fibrosis is the usual response so that fibro-caseous disease and a tendency to consequent cavity formation is the finding in the majority of patients. We do not know if such usual adult manifestations are really a secondary or tertiary stage of the disease because we have no mass X ray observations under the age of seventeen or eighteen. Similarly we do not know what really happens to a primary infection in adolescence or adult life. Recent experience suggests that a young adult who is negative to tuberculin and gets first contact with an open case of tuberculosis shows film findings exactly like those which can accompany an erythema nodosum. There is a large confused hilar shadow often with scattered indistinct pulmonary shadows. These latter may be areas of lymphangitis and

consequent collapse of bunches of alveoli. Some are complicated by pleural effusion. It is not uncommon to recover tubercle bacilli from the stomach washings. The X-ray such as shown in Fig. 95



FIG. 95. The X-ray appearances which can accompany erythema nodosum.

is usually read as Sarcoidosis of lung. The writer has followed some such cases by serial films through stages of fleeting shadows in the both apices and first infracavicular deposit with positive sputum within eighteen months of the appearance of erythema nodosum.

In the involvement of broncho-pulmonary segments the track is again through the lymphatic system there is no change in the fundamental pathogenesis of the disease. Our studies on the anatomical relations of the alveoli reminded us that they connect with the lymphatics through lymph spaces which surround the lobules. It is into these spaces that the phagocytes containing the tubercle bacilli now enter after being caught in the lymphatics which accompany the dorsal bronchioles and the first reaction is a cellular exudate in the alveoli from irritation of their walls. This is a non-specific inflammatory oedema the same reaction which we saw in epituberculosis.

is not uncommon to find the first recognizable foci of adult tubercle as rounded areas of homogeneous loss of translucency in the inner or outer thirds of the right infra-clavicular region. They are round because as we have noted before the cone of infected parenchyma is photographed through its long axis. They may be in small and multiple areas up to about $\frac{1}{4}$ inch in size when first noted but are usually single and about three times this size. They are then known as Aschmann's foci. They are then fairly well defined, and entirely homogeneous. The apparent striations on them are really the normal markings of lung tissue lying anterior to them on the postero-anterior film as can be shown by taking either an antero-posterior film which presents them nearer to the surface and therefore to the film or by doing a tomograph (see Fig. 46).

There is one reaction to the focus that is quite common in the upper lobe especially the right and is often called erroneously 'tuberculous pneumonia'. We saw that in *epituberculosis* there is a possibility that lymphatics round a bronchus can be so swollen by reaction that they occlude the lumen and produce a localized area of collapse as a complication. A comparable reaction can take place in response to an acute Aschmann's focus. Swelling in the lymph-spaces together with much catarrhal exudate in irritated alveoli round them can give a heavy shadow part of which is due to actual lobular collapse. The interlobar fissure is drawn up and the mediastinum well displaced to the affected side. The lymphatic inflammation is quickly passed to the interlobar fissure which is always heaviest in its shadow just below the lesion. This is the condition that was compared for differential diagnosis with upper lobe atelectasis (see Fig. 76).

Rest and serial films will often show the true state of affairs just as the catarrhal reaction often disappears round foci in other parts of the parenchyma now both the catarrh and the lymphangitis can be seen on serial films to retrogress to more and more translucency while in the rounded area of the causative deposit we not infrequently see a rapidly forming cavity with a fluid level. The condition has been therefore but a manifestation of the severity of the attack.

The temperature and constitutional disturbance may simulate pneumonia at onset but the dullness of the upper lobe is more posterior than anterior and we shall find signs of mediastinal displacement which as we have seen can occur only in complications of pneumococcal pneumonia. Later the film can simulate

abscess formation in a lobe collapsed by bronchial carcinoma but as the cavitation develops in the tuberculous case the rest of the lobe clears above the lesser fissure whereas in true carcinomatous atelectasis the surrounding density remains. By this time sputum examinations will generally clinch the diagnosis.

The large rounded form of the Asmann's focus as we have seen by no means a necessity. Individual acini may be seen on the film as tiny individual dot like foci congregated together and giving no evidence of inflammatory exudate. We have already noted that such single or multiple foci can be concealed by overlapping bony shadows. Where there is any doubt the rib intersections must be studied very closely and an antero-posterior view taken. All this means is that there is less acute invasion or that we are looking at a later stage when the exudate has gone. It does not mean that these areas will not go on to the same after-effects. Serial films show they can proceed to cavitation of slower onset although they are on the whole more inclined to go on to organization and fibrosis without much drag on the bronchus. If however they create and affect by extension other acini in their immediate neighbourhood they are no different in their film findings from those Asmann foci which appear to break up into their constituent parts and produce a picture of small triangular areas that merge into the shadow of the inflamed lymphatics round congested bronchi tracking towards the hilum. The disease is following its constant pathogenesis. It is attempting to drain by the lymphatics to the hilar glands.

This type of individual acinar deposit is common in the supra-clavicular apex and means that the posterior apical branch of the upper lobe bronchus has been affected (see Fig. 3). Experience with mass radiography shows this is far commoner site of disease than was formerly believed and many cases that show active Asmann foci below the collar bone have evidence of previous true apical deposits. Indeed this is so common that it appears probable that infra-clavicular tubercle is a second or perhaps a secondary attack in previous and unsuspected more peripheral disease never quite healed. Whether such true apical tubercle is an outpost of the primary focus of childhood that remains dormant until adolescence or may by the modern possibilities of serial film examination soon be able to state.

It can be extremely difficult to decide whether such deposits are present in the supra-clavicular apex. On rapid

shadows the shadow of the sterno-mastoid pleural tenting and pleural striæ can all give confusing shadows. The antero-posterior view is often of great help in the final opinion.

If we now looked at the cut surface of the post mortem specimen we should see a collection of tiny nodules of collapsed and organized acini each round a central more or less occluded, bronchiole. We might also recognize many white strands due to fibrosis of the peribronchial and perivascular connective tissue the latter narrowing the blood vessels and helping to contribute to the endarteritis obliterans which is a constant feature of the disease. The hilar glands are hardly if at all affected the fibrosis has allowed but few bacilli to reach them.

It is little wonder that tuberculosis is without symptoms or gross physical signs especially to stethoscope for a considerable time after its establishment in adult form. It does produce lack of movement however and very soon afterwards destruction of elastic tissue begins by organization and the results of bronchial blockage. The sterno-mastoid sign reflects drag on the mediastinum, but only careful stethoscopic listening will demonstrate the first sound of the material evacuating from the lobules into their supplying bronchioles. We shall then hear evidence of the bronchiole contents in fine sibilant at the very end of inspiration increasing in intensity to reach a crescendo at the very end. Remembering

our stethoscope findings in bronchitis we recognize a distinctive and diagnostic difference in the position in inspiration of this sound which is exactly the same in its quality to the ear in both diseases. In bronchitis we heard it at the beginning of inspiration, lessening with the increasing depth of breathing, and connoting obstructive material consisting of breaking-

down elements of the wall what we hear in phthisis is material evacuating into the terminal distal ends of the bronchioles from their connecting lobules. With these sibilant or coarser rhonchi we shall soon hear the dry crepitations of fibrosis also most marked at the end of inspiration and not disappearing with cough; these are the signs of the peribronchial fibrosis. In other words

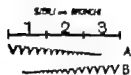


FIG. 98.

- A. Bronchitis.
B. Pulmonary tuberculosis.

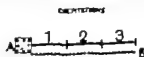


FIG. 99.

- A. With act of cough in pleural thickening.
B. With fibrosis of lung.

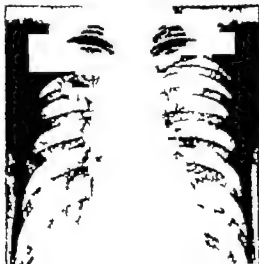
we have stethoscopic evidence of infiltration and fibrosis

Thickening of the pleura overlying the broncho-pulmonary segment follows very soon. Its stethoscopic sign we shall also find, in a burst of fine dry crepitations with the act of cough which disturbs the fine adhesions of organizing exudate between the visceral and parietal layers

The progress of a focus that keeps its rounded form for some time shows by serial films that it may although rarely seem to disappear altogether. It may split as already noted into small individual lesions or go on to a drawn out mass of fibrotic strands and organized areas. Sometimes there will be rapid cavitation with occasionally a fluid level at first X ray recognition. Usually however the cavity is due to a slow weakening of the walls which are dragged peripherally in ragged fashion by organization here and collapse there so that the film shows an area that has lost in great part its rounded formation and is merging into the shadow



FIG. 100. Cavitation in pulmonary tuberculosis.



of the larger bronchi now outlined by surrounding thickening connective tissue as they track downwards and inwards to the hilum.

Critical inspection of the film of an established case with activity gives us a lesson in living pathology. If we look at it closely we see it has a background of fine generalized loss of translucency, this is thickened plastic pleurisy. At the edges of the lesion we see areas of slightly greater loss of translucency, they are due to the catarrhal exudate in irritated lobules in the process of invasion, i. e. the areas of advancing infiltration merging into the heavier shadows of lobules now containing tubercle bacilli that have penetrated their walls and are producing caseation. More centrally we see still denser individual lesions, clear cut, standing out on distant viewing like the thickened bronchial walls, because they have round them lobules distended by complementary emphysema. These denser areas of collapsed and organized lobules are fused and solid in clumps, round the dilated and destroyed bronchial wall to form the heavy but broken up outline of cavity. The picture is distinctive and diagnostic unlike any other in chest disease.

There is possible confusion with the cavity of true lung abscess but, as we have already seen, the cavity surround in this case looks

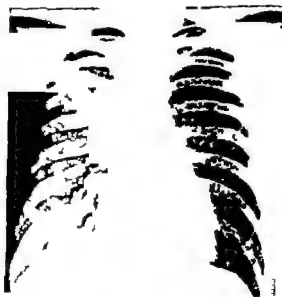


FIG. 101 Same case as in Fig. 102. A-P view shows cavity is present.

as if all the affected lobules had been suddenly frozen *in situ* to form a continuous band with no areas of intervening lobular emphysema and therefore not altering essentially in their detail on distant as against close viewing. We saw also that a further and important point in such differential diagnosis is that the chronic tuberculous cavity is seldom the only evidence of phthisis. It is much more often in a lobe throughout which can be seen scattered areas of infiltration with fibrosis.



FIG. 103. Tuberculous cavity.

The advent of the cavity gives its own adventitious sounds. The presence or absence of whispering pectoriloquy is not a reliable diagnostic finding, the more the contents of the cavity at the time of listening, the less the pectoriloquy. A much more constant

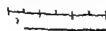


FIG. 104. Metallic rales in tuberculous cavities.

sign is the presence of coarse metallic rales. They are of quality exactly as those we heard in bronchiectasis and in lung abscess, but they are now heard beginning in the second half of inspiration and become

more and more insistent with the depth of breathing, until they reach a crescendo at the very end.

We must remind ourselves that considerable portions of lung tissue are involved before added sounds are heard. It is a fairly safe rule to conclude that signs heard above the clavicle mean the upper third of the lung is involved and signs below the clavicle mean at least half of the lung is involved. An example of the influence of other factors in their production is that where laryngeal tubercle is present we can consider the extent three times that indicated by our stethoscope findings.

Acute Exudative Phthisis of Adolescence and Adult Life

This type of pulmonary tuberculosis is fortunately nowadays rare in this country as acquired immunity is widespread. We do however occasionally see it as a steadily advancing disease unrestrained by any fibrosis. Inflammatory exudate in the lobules is rapidly replaced by caseating material which on evacuation fills the bronchi whole areas breaking down to be coughed up and to leave in the lung fields ragged vacancies which are therefore cavities of entirely different mode of formation from those we have already considered. As no barrier is placed in the way of lymphatic drainage tubercle bacilli flood the hilar glands and they in turn go on to rapid caseation.

The film therefore shows many areas of translucency among widespread homogeneous densities. The translucencies are the cavities often of no clear definition but occasionally looking like ticket punchholes where there has been a sudden evacuation through a bronchiole of rapidly caseated tuberculous material round it. Bunches of heavily infiltrated lobules then make up what is only an apparently confining wall. Hilar shadows are heavy indistinct and confused made up of the caseating glands surrounded by much swelling in lymphatics and much increased hyperemia.

Movement of the chest is poor there is generalized loss of note with no offsetting areas of emphysema. The predominant part of the affected zones of the lungs is silent, as residual air is replaced by exudation and caseation but now and again we hear coarse rhonchi caused by the bronchial obstruction. These are especially noted being exaggerated and insistent even after cough in areas going on to cavity formation by rapid evacuation. We are listening to tuberculous bronchitis which is really a flooding of the bronchi by the evacuating caseated tuberculous deposits.

CHAPTER XIX

PLEURISY

THERE are three main forms of pleurisy fibrinous dry sero-fibrinous, or pleurisy with effusion and purulent or empyema. There is no hard and fast delimitation between the forms, seeing that there is always some slight fluid present with the first while the second and third really vary only in the amount of pus in their cellular content. All three forms can be caused by the pneumococcus the streptococcus or the tubercle bacillus.

Dry pleurisy may be primary to the pleura by the presence in its layers of the bacillus of Koch. It is however almost always secondary to pulmonary tuberculosis if it be tuberculous even if it is actually found in the parietal layer in which case what has happened is that bacilli from a previous frank effusion have penetrated this layer to its intimal coat. Dry pleurisy is also found with pneumonia and broncho-pneumonia and their suppurative complications and may be diaphragmatic in site when secondary to abdominal disease which is commonly sub-phrenic abscess.

Its effect on the membranes depends on its cellular content the higher the polymorph percentage-count the less being the chances of ultimate and permanent damage. This is why lobar pneumonia, while producing a heavy exudate on the visceral layer yet leaves little ultimate damage in the vast majority of cases as its greyish-yellow thick deposit even should it undergo some organization will be removed eventually by the proteolytic enzymes of its preponderant polymorph content. On the other hand tuberculosis produces an exudate which contains a high percentage of fibrin as against polymorphs and so is much more liable to proceed to permanent adhesion between layers of the pleura over parts of the lung affected by phthisis.

We can follow these effects of dry pleurisy on the film. We see much more density from recent pneumonic than from a recent secondary tuberculous pleurisy but the pneumonic one followed in serial films will almost always disappear while the tuberculous one leaves behind haziness of fine homogeneous

type to act as a background to those lung shadows of infiltration and fibrosis which are the underlying cause of its appearance.

There are two exceptions to this rule the first being demonstrated in so-called tenting of the diaphragm. We saw already that localized lung affections of pneumonic type are slow of disappearance and incline to healing by repair rather than by resolution as against their more generalized forms. The same effect is seen in interlobar pleural infections. We have noted earlier that very often a localized tent shaped shadow is seen on a film about the mid point of the diaphragm on one side more often the right than the left.



FIG. 103. Filling of costo-phrenic angle plus lung focus; the cause is tuberculous.

We noted the second when discussing the diaphragm. It consists in that filling-in of the extreme costo-phrenic angle by a shadow which has no clinical significance unless it be accompanied by a lung focus. Without evidence of such a focus it should never be read as tuberculous in origin. It is almost always due only to a previous known or unrecognized pleural reaction of pneumonic or broncho-pneumonic origin.

The most painful type of dry pleurisy is the pneumonic. The progress of the average secondary pleurisy whatever its cause is from locral to parietal layer each being involved first by inflammatory reaction and then by the specific reaction of the penetration of the causative organism, in the same way as we saw the progression of any lung disease involving the alveoli from catarrhal to specific exudation. The pneumococcus first irritates and then invades the visceral pleura as it reaches the periphery of the lung sector. It seldom does more than inflame the parietal layer. (Edema stretches and makes this layer supersensit. Dragging by adhesion between the moving visceral and the static parietal layer then produces great pain. With lung abscess pain is not frequent and with plastic pleurisy of tuberculous origin the patient complains more of a rheumatic ache than an acute discomfort unless as in pneumonia he has a widespread reaction to the parietal layer. In which case he is almost always about to develop frank effusion.

As already noted sero-fibrinous effusion differs from dry pleurisy only by virtue of the fact that fluid is in amount sufficient for physical or usual demonstration. The cause in most cases seen in general practice is tuberculous of the lung. It is to this latter type that we shall confine our detailed description of frank pleurisy.

The fluid contains much fibrin & high percentage of protein and a preponderance of lymphocytes as against polymorphs when compared with the high polymorph content of the pneumococcal and the rarer streptococcal effusions. It is usually clear and has an opalescence due to suspended fine particles of fibrin easily seen in fluid examined *in situ* through a thoracoscope and quickly forming the typical clot in samples withdrawn for visual and laboratory examination. It is not cloudy like the pneumonic exudate with its polymorphs tending to form pus. The fibrinous content less the dangerous liability to the formation of thick coverings on the visceral and parietal layers when the fluid is left to organize in the pleural cavity. Tubercle bacilli seldom appear in smears and may fail to appear in culture if the sample is examined under few days from the onset of the effusion. Infection has not yet followed irritation and consequent inflammatory exudation. Failure to realize and act on this simple fact of pathogenesis may have serious consequences for the patient by inefficient treatment founded on false

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In one laboratory examination report which has been made too soon and without clinical notes to the pathologist

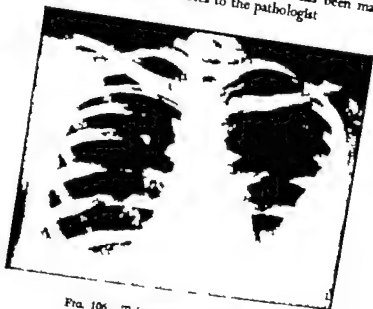


FIG. 106. Tuberculous Pleural Effusion.

To physical examination there will be dullness beginning in the lower axilla and extending upwards with the increase in effusion. While there will be silence in the area displacing the lung, there will naturally be increased breath sounds over that part of the lung on the limits of the fluid which are by it pressed inwards and upwards. The collapsed lung acts as a sounding-board. The outstanding point is however the displacement of the mediastinum to the opposite side demonstrated in a positive sterno-mastoid on that side and usually supported by evident displacement of the apex beat to palpation and auscultation.

We may remind ourselves how the lung reacts to the presence of fluid in the pleural cavity. When the negative intra-pleural pressure is brought nearer to that of the atmosphere there will be less outward pull on the underlying lung. Its elasticity will come into play therefore and draw it inwards to the anchorage of elastic tissue at the hilum. From the first the reduction of the pleural pressure on the affected side will make that on the opposite side greater in comparison and so the mediastinum will be first

of all drawn to this opposite side. With more fluid the pressure in the pleural cavity will be greater than that of the atmosphere, and so begin to bear in on the parenchyma that is is it greater than the pressure of the residual air in the lobules. But we can see that there does not need to be any actual push from the affected side in order to give a positive sterno-mastoid on the other.

After this point the reaction of the lung and the mediastinum depends on whether they are healthy or already diseased. A lung with preceding fibrosis will naturally resist collapse and will move bodily against the mediastinum, while a fixed mediastinum e.g. one that has been affected by previous pleurisy will resist displacement. What this means is that we must consider all these separate factors and not conclude that the amount of mediastinal displacement is in anything like exact ratio to the amount of fluid or the actual change in pressure in the intra pleural space.

While it must be stressed that no pleural effusion has been fully investigated unless exploratory puncture for visual and laboratory diagnosis and blood film examination, have been carried out. It is none the less true that tuberculous exudate has characteristic film finding. Unfortunately this cannot be demonstrated by the print of the film as it can by looking at the film itself on the viewing box. In its early days it has a specific fine loss of translucency through which the ribs can be seen almost as clearly as they would be with the presence of a pneumothorax. Towards the mid zone from above downwards there is a comparative density conceals inwards running from the axilla to the diaphragm, but this is not in the uncomplicated case clear single line of demarcation. It is made by the combined shadow of fluid and lung, the former lying to some depth laterally both in front and behind the latter. If there is no underlying lung disease we ought to be able to see exaggeration of the normal vessel markings to the inner side of this junction, owing to the hyperemia in the lung contracted towards the hilum. If these markings cannot be seen we must suspect consolidation from for example pneumonia.



FIG. 107 Tuberculous pleural effusion.

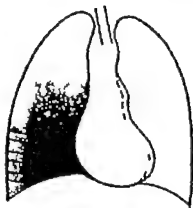


FIG. 103. Pneumonia right lower lobe, with fluid. Note loss of vascular markings, as in uncomplicated pneumonia, but displacement of mediastinum due to complicating effusion.

The picture is therefore not like that of uncomplicated lobar pneumonia of the lower lobe with its density at the base decreasing from the diaphragm upwards towards the lingula and equal at any one level between the axilla and the mediastinum. In pleurisy there is increasing loss of translucency from the axilla inwards to the junction of fluid and lung. Moreover it will be differentiated also by the position of the heart and the mediastinum for we saw in our study of the pneumonias that they do not cause displacement.

If fluid has been present for some time before the film has been taken the concave density may have assumed a sharp outline. This is because organization of the fibrinous exudate on the visceral pleura has already taken place and the patient is thereby in danger of incomplete re-expansion of the lung even with immediate and complete withdrawal of fluid. Should the effusion be of still longer standing, organization of the fibrin floccules gives a dense white shadow occupying the area between the axilla and the now sharply defined border of the lung fixed and compressed.

The shadow is not now anywise different from that of true empyema although the latter in its acute stage will cause displacement to the opposite side. This will be a definite aid in visual diagnosis but it must call for the accessory aids of blood and exploratory-puncture examination. The usual cause in general practice cases is the pneumococcus for true tuberculous empyema is a rarity and seldom met with except as a complication of a previous clear effusion especially if such effusion has occurred as a hydro-pneumothorax.

The average case of such idiopathic pleurisy as just described clears completely with no remaining evidence in pleura or lung. If we see on the film some filling of the costo-phrenic angle and no lung focus is evident on a postero-anterior film we ought to take lateral and lordotic films and even tomographs if we wish to

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be as certain as is humanly possible of our final opinion. Often the small tuberculous deposit is in the periphery in the upper mid zone, or in the supra-clavicular apex from involvement of the posterior apical bronchus.

Interlobar effusions apart from that in the lesser fissure on the right side are seldom evident or demonstrated to their true extent in the postero-anterior film. A lateral shows them clearly forcing apart the interlobar septum and usually bow shaped because they are pressing on the parenchyma of both lobes (see Fig 8). Fluid in the main fissure can give on the postero-anterior film shadow very like that of true diaphragmatic effusion. If emphysema secondary to abdominal disease is sub-phrenic abscess. Both sit over the diaphragm and are inclined to follow its outline. The physical signs however will help us to differentiate because while the latter gives us dullness both anteriorly and posteriorly the dullness due to interlobar effusion is especially marked in front. A lordotic picture tells us too for the true interlobar effusion rises through the middle of the lung field like drawn-out tent sitting on the diaphragm.

The usual dry diaphragmatic pleurisy shows no evidence in film examination to account for the acute shoulder and upper arm pain that accompanies it.

Pneumothorax

From what has been said of the effects of pleural effusion on lung elasticity and mediastinal shift we can visualize the changes in pneumothorax without further detailing them. The effect on the film findings of the underlying lung is the same and the diagnosis most rest on the alterations noted both within and without the lung field. As against pleural effusion we now have complete absence of shadow between the rib interspaces outside the line of the retracted lung, no loss of translucence and no lung markings are evident (see Fig 2).

In the simple or non-tuberculous case this is extremely rare so fluid to appear in the pleural space and it may be possible to see on the edge of the collapsed lung emphysematous bullae one of which has burst to produce the pneumothorax. In the case where pneumothorax complicates an already demonstrable or undemonstrable lung tuberculosis fluid is exceedingly common.

The commonest abnormality of mediastinal glands is of course due to tuberculosis and is dealt with in the chapter on Childhood Tuberculosis

Lymphadenoma Hodgkin's Disease

This condition usually appears first on the postero-anterior film as a moderately sized shadow in the upper mediastinum. It is slightly more dense than the shadow of the superior vena cava which it generally covers. This is because it is almost always first evident



FIG. 109. Lymphadenoma.

in the right para-tracheal glands even if it has been bilateral from the beginning. Its outer border is clear-cut but in a series of bulges as against the more continuous sweep of the rounded or oval

outline of other abnormal shadows in the same position. Its lower border and the greatest abnormal width of the mediastinum lie



FIG. 110. Lymphadenoma. Oblique view of case in Fig. 95.

above the upper aortic shadow. Serial films show long increase in size across the mediastinum thus displacing the trachea and the oesophagus and into the neck. Such spread may obscure the involvement of the groups of glands although they are sometimes be identified on hard postero-anterior and ro-posterior or lateral views. If spread includes the pulmonary tracheo-bronchial and hilar groups the picture is one of overlapping egg-like shadows along an indefinite upper cardiac border. We may now see pulmonary involvement in streaks spreading from the outer border along the lines of the bronchi as definite nodules or as homogeneous irregular shaped patches some with the density of pneumonia and some perhaps edged with areas of ground-glass appearance because they are causing collapse of surrounding alveoli.

At this stage we may need every accessory aid including irradiation to which it responds less to distinguish this disease from lympho-sarcoma



FIG. 111 Lymphosarcoma.

Lympho-sarcoma

This is the next commonest abnormality of glandular origin. It is generally bilateral from its onset. In early stages its density is comparable to that of lymphadenoma but it is generally more rounded in its right border. It grows more rapidly to produce marked broadening of the superior mediastinum and may so obscure the aortic shadow that it cannot be diagnosed against aortic aneurysm.

Dermoid

The oval homogeneous dermoid of pericardial inner side and gives a



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num on its
'See Fig. 49)

On the postero-anterior film it is usually situated lower than the level of the aortic arch. We may require an artificial pneumothorax to be certain of its connection with the mediastinum.

When a dermoid arises from embryonic remnants of the thymus the oblique fissure will show it lying in the anterior superior mediastinum. It does not displace the trachea or the oesophagus. We have already noted that film examination alone will not diagnose

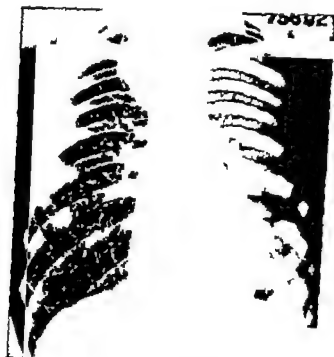


FIG. 112. Dermoid of lung

this tumour from large central neurofibroma. From the crurae of the diaphragm the same are rare. Only very rarely does a dermoid originate from the parenchyma.

Neuro-fibroma

This the commonest benign chest tumour arises from an intercostal nerve. It is usually discovered by chance during mass radiography or screening. Its loss of translucency is comparable



F 113 Neurofibroma.

in homogeneity and density with lymphadenoma but it is more rounded in its outer edge. Antero-posterior, postero-anterior, lateral and oblique films will demonstrate it as lying in the posterior superior mediastinum. A better Bucky film of the spine may show its connection with the nerve. It is soft in structure and slow in growth and therefore shapes itself against the resistance of surrounding structures hence its usually clear-cut and rounded outer edge on the postero-anterior film. It does not displace the trachea.

the oesophagus. Occasionally it develops in a central lung position when as noted by pneumothorax may be necessary for differential diagnosis against dermoid and hydatid cyst.



FIG. 114 Neurofibroma, lateral view

Substernal Thyroid

A substernal thyroid gives a triangular homogeneous shadow in the upper anterior mediastinum with the base of the triangle at the thoracic outlet and the apex pointing downwards. It may be more pronounced on one side or be completely to one side of the midline but usually depresses the main vessels and pushes the aorta to the left so that the aortic knob is prominent. Very often it presses backward on the trachea. Screening will show it rises when the patient swallows.

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Lobar Carcinoma

Other parenchymatous growths have been considered in the chapter on Round Shadows.

Primary sarcoma of the lung is rare but primary carcinoma is common. It usually begins in a large bronchus and therefore produces lobar collapse first by increasing obstruction and later by actual alveolar involvement in the manner described in the chapter on atelectasis.

There we noted its tendency to cast a shadow which is densest at the hilum. Hard films may show this density to be made of two shadows: a very dense one of enlarged glands within another slightly less dense one of collapsed lung.

Loss of lung volume is shown by the falling-in of the rib-spaces outside the affected area and by the drift of the mediastinum and the heart to the same side. We saw why this drift is more marked in lower lobe than in upper lobe atelectasis; not only is there more loss of volume but the heart is much more readily displaced from normal in its lower and heavier half. The swing from this fixed position of drift still further to the affected side on inspiration and the return on expiration so easily seen on the screen is also much more marked with lower lobe and complete lung collapse than with upper lobe collapse. Pulmonary fibrosis or pleural obliteration do not produce this swing. We have already seen how we can get further aid in the diagnosis of collapse against fibrosis if we note the positions of the oesophagus and the trachea.

Nerve involvement is quite common in upper lobe carcinoma as the recurrent laryngeal on the left side and the phrenic nerve on the right side can very easily become embedded and compressed in masses of enlarged glands in the upper mediastinum. Extension of a right upper lobe carcinoma can therefore produce an increasing rise of the right diaphragm until complete paralysis of the nerve brings complete immobility of the diaphragm in the position of complete expiration equivalent to that seen with excision of the nerve. The conjunction of a raised diaphragm and an atelectatic right upper lobe is diagnostic of right upper lobe carcinoma.

Screening will give us still further aid. The movement of the diaphragm is paradoxical. This abnormal movement occurs also in lower lobe carcinoma. It cannot be seen on the right side since the shadow of the muscle is merged with the liver shadow.

but if the patient is given a Siedlitz powder it is possible to demonstrate it on the left side unless there is a large pleural effusion.

When collapse of the lower lobe by carcinoma has reached what we described as the third complete stage we may require lipiodol injection for diagnosis against foreign body or bronchiectatic collapse. In both these conditions we are almost certain to find some lipiodol in the collapsed lobe.

Atelectasis of the whole lung occurs when the growth is in a main bronchus. We find a homogeneous loss of translucency all over one side with a density varying from that of pleural effusion to that of empyema. The following points will lead us to arrive at the true diagnosis. The rib-spaces will be flattened on the side of the shadow. The mediastinum and heart will be displaced to this side and wing still further over with each respiration. The oesophagus will lean over and the trachea will be displaced well



FIG. 115. Carcinoma of the lung—nodular (nodal) form.

over. The value of physical examination in the differential diagnosis of collapse with complicating effusion has been mentioned.

The appearance and differential diagnosis of the type of nodular carcinoma which throws a single rounded shadow has been discussed in the chapter on Rounded Shadows. Three other main forms of nodular carcinoma remain to be mentioned—mediastinal, medial and basal. The mediastinal or hilar form originates near the bifurcation of the trachea. Instead of blocking the lumen it erodes the wall and as it involves the glands and all the mediastinal tissues produces a shadow which is large, rounded, homogeneous and extremely dense. Its edge may be well-defined or ragged. If it is ragged it can usually be seen to send out irregular, fibrotic-like threads of differing size and shape into surrounding lung tissue.

The medial form is due to carcinoma of a main bronchus close to its origin. The shadow looks as if it were part of the main cardiac shadow, resting like a hump on the entricular border. The outline may be sharp or quite irregular. The shadow changes very little to serial examinations over a period of time.

The basal form gives an ill-defined lung shadow attached to the hilum by many irregular streaks. It is usually of varying densities as it has within it areas of obstruction of bronchioles. Very often it proceeds to the usual picture of lower lobe complete collapse.

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